Foley, S
09/702498

-key terms
Genomic RNA

(FILE 'CAPLUS' ENTERED AT 11:50:27 ON 24 MAY 2001)
L1 137 SEA FILE=CAPLUS ABB=ON PLU=ON ((SHIPPING FEVER OR SENDAI OR BOVINE(W)(PARAINFLUENZ? OR PARA INFLUENZ?) OR HAEMAGGLUTIN? OR HEMAGGLUTIN? OR PARAMYXO OR PARA MYXO)(W)VIR? OR PARAMYXOVIR? OR PARA MYXOVIR? OR HVJ)(S)VECTOR
L10 8 SEA FILE=CAPLUS ABB=ON PLU=ON L1 AND ((RNA OR RIBONUCLEIC OR RIBO NUCLEIC)(5A)GENOM?)

L10 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:229785 CAPLUS
TITLE: Safety evaluation of hemagglutinating virus of Japan-artificial viral envelope liposomes in nonhuman primates
AUTHOR(S): Tsuboniwa, Naoki; Morishita, Ryuichi; Hirano, Tadamichi; Fujimoto, Jiro; Furukawa, Shigenori; Kikumori, Mikito; Okuyama, Akihiko; Kaneda, Yasufumi
CORPORATE SOURCE: Division of Gene Therapy Science, Graduate School of Medicine, Department of Specific Organ Regulation, Osaka University, Osaka, 565-0871, Japan
SOURCE: Hum. Gene Ther. (2001), 12(5), 469-487
CODEN: HGTHE3; ISSN: 1043-0342
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We tested, in cynomolgus monkeys, the safety and effectiveness of a hybrid liposome **vector**, **hemagglutinating virus** of Japan (**HVJ**)-artificial viral envelope (AVE) liposomes, for human therapeutic gene transfer in a series of expts. In a repetitive i.m. administration study, vehicle control macaques (n = 2), which were treated with HVJ-AVE liposome suspension, received repetitive i.m. injections of 2 mL of test substance. Human hepatocyte growth factor (HGF) cDNA-inserted expression **vector** (pUC-SR.alpha./HGF) injection animals (n = 2), which were treated with **HVJ**-AVE liposome suspension contg. pUC-SR.alpha./HGF, received repetitive i.m. injection of 2 mL of test substance. General body condition, hematol., blood chem., and serum HGF were detd. sequentially before treatment and 7, 21, 28, and 29 days after treatment. Elevations in HGF were detected in monkeys injected with pUC-SR.alpha./HGF. After this observation period, macaques were killed for autopsy and histol. examn. PUC-SR.alpha./HGF was detected by polymerase chain reaction (PCR) anal. in the liver, spleen, and at the injection site. In single i.v. administration study, control macaques (n = 4) received a single i.v. injection of 10 mL of physiol. saline. Vehicle control animals (n = 5) received a single i.v. injection of 10 mL of HVJ-AVE

liposome suspension. DNA-treated animals (n = 7) received a single i.v. injection of 10 mL of HVJ-AVE liposome suspension contg. plasmid DNA [pcDNA 3.1(+)]. General body condition, body wt., hematol., blood chem., and urine compn. were detd. sequentially before treatment and 1, 14, 21, and 28 days after treatment. After this observation period, macaques were killed for autopsy and histol. examn. PcDNA 3.1(+) was detected by PCR anal. on day 1 in lung, liver, and spleen of all monkeys, in kidney of one of two monkeys, and in heart of one of two monkeys. However, no DNA was detected in any of the tissues examd. on days 14, 21, and 28. No virus **genomic RNA** was detected by reverse transcription (RT)-PCR anal. with HVJ-specific primers. In this series of safety evaluations, the animals tolerated the safety study with no change in body wt. or general condition. No hematol. changes or alterations in blood chem. or urine compn. was detected. Moreover, no histol. changes were obsd. This safety evaluation study demonstrates the safety, feasibility, and therapeutic potential of the novel transfection vehicle, HVJ-AVE liposomes, in humans.

REFERENCE COUNT: 36
REFERENCE(S):
(1) Allen, T; Proc Natl Acad Sci U S A 1988, V85, P8067 CAPLUS
(2) Alving, C; Proc Natl Acad Sci U S A 1978, V75, P2959 CAPLUS
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(6) Hayashi, S; Biochem Biophys Res Commun 1996, V220, P539 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:466593 CAPLUS
DOCUMENT NUMBER: 133:345261
TITLE: A cytoplasmic RNA **vector** derived from nontransmissible **sendai virus** with efficient gene transfer and expression
AUTHOR(S): Li, Hai-Ou; Zhu, Ya-Feng; Asakawa, Makoto; Kuma, Hidekazu; Hirata, Takahiro; Ueda, Yasuji; Lee, Yun-Sik; Fukumura, Masayuki; Iida, Akihiro; Kato, Atsushi; Nagai, Yoshiyuki; Hasegawa, Mamoru
CORPORATE SOURCE: DNAVEC Research Inc., Tsukuba, 305-0856, Japan
SOURCE: J. Virol. (2000), 74(14), 6564-6569
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We have recovered a virion from defective cDNA of Sendai virus (SeV) that is capable of self-replication but incapable of transmissible-virion prodn. This virion delivers and expresses foreign genes in infected cells, and this is the first report of a gene expression **vector** derived from a defective viral genome of the **Paramyxoviridae**. First, functional ribonucleoprotein complexes (RNPs) were recovered from SeV cloned cDNA defective in the F (envelope fusion protein) gene, in the presence of plasmids expressing nucleocapsid protein and viral RNA polymerase. Then the RNPs were transfected to the cells inducibly expressing F protein. Virion-like particles thus obtained had a titer of 0.5 .times. 108 to 1.0 .times. 108 cell infectious units/mL and contained F-defective **RNA genome**. This defective vector amplified specifically in an F-expressing packaging cell line in a trypsin-dependent manner but did not spread to F-nonexpressing cells. This vector infected and expressed an enhanced green fluorescent protein reporter gene in various types of animal and human cells, including nondividing cells, with high efficiency. These results suggest that this vector has great potential for use in human gene therapy and vaccine delivery systems.

REFERENCE COUNT: 36

REFERENCE(S):
(1) Arai, T; J Virol 1998, V72, P1115 CAPLUS
(3) Bukreyev, A; J Virol 1996, V70, P6634 CAPLUS
(4) Cathomen, T; EMBO J 1998, V17, P3899 CAPLUS
(5) Collins, P; Proc Natl Acad Sci USA 1991, V88, P9663 CAPLUS
(6) Conzelmann, K; Annu Rev Genet 1998, V32, P123 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:355373 CAPLUS
DOCUMENT NUMBER: 133:39815

TITLE: Analysis of RNA polymerase activity associated with NP protein of paramyxovirus

AUTHOR(S): Masago, Akinori
CORPORATE SOURCE: Dep. Neurovirol., Res. Inst. Microbial Dis., Osaka Univ., Japan

SOURCE: Osaka Daigaku Igaku Zasshi (2000), 52(3-4), 125-128
CODEN: ODIZAK; ISSN: 0369-710X
PUBLISHER: Osaka Daigaku Igakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB During the life cycle of Sendai virus (Se V), three types of viral **RNA** (mRNA, full-length **genome** and antigenome **RNA**) are synthesized in the cytoplasm by the viral

nucleocapsid proteins (NP, P and L). Although L protein or the complex of P and L proteins have been assigned to viral RNA polymerase which utilize the NP protein-RNA complex as a template, detail mechanism of the viral RNA synthesis is still obscure. In this study, we established a cell line expressing T7 RNA polymerase stably (LLCMK2#T7) and investigated a function of NP protein of SeV. We constructed a SeV mini-genome (pHVLuciB), which directed the synthesis of (-) strand **RNA** consisting of SeV **genome** 3' leader-luciferase-SeV 5' trailer under T7 RNA polymerase promoter. When pHVLuciB was transfected to LLCMK2#T7 cells together with pGEMNP (NP protein expression vector), luciferase activity and (+) strand luciferase RNA were detected in the transfected cells. The (+)strand RNA synthesis required the expression of full-sized NP protein and the (-) strand template with genome 3' leader. These results suggest that SeV NP protein has intrinsic RNA polymerase activity that directs the synthesis of (+) strand RNA.

L10 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:483920 CAPLUS
DOCUMENT NUMBER: 131:282150
TITLE: Accommodation of foreign genes into the Sendai virus genome: sizes of inserted genes and viral replication
AUTHOR(S): Sakai, Yuko; Kiyotani, Katsuhiro; Fukumura, Masayuki; Asakawa, Makoto; Kato, Atsushi; Shioda, Tatsuo; Yoshida, Tetsuya; Tanaka, Akemi; Hasegawa, Mamoru; Nagai, Yoshiyuki
CORPORATE SOURCE: Institute of Medical Science, Department of Viral Infection, University of Tokyo, Tokyo, Japan
SOURCE: FEBS Lett. (1999), 456(2), 221-226
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Sendai virus (SeV) is an enveloped virus with a neg. sense **genome RNA** of about 15.3 kb. We previously established a system to recover an infectious virus entirely from SeV cDNA and illustrated the feasibility of using SeV as a novel expression vector. Here, we have attempted to insert a series of foreign genes into SeV of different lengths to learn how far SeV can accommodate extra genes and how the length of inserted genes affects viral replication in cells cultured in vitro and in the natural host, mice. We show that a gene up to 3.2 kb can be inserted and efficiently expressed and that the replication speed as well as the final virus titers in cell culture are proportionally reduced as the inserted gene length increases. In vivo, such a size-dependent

effect was not very clear but a remarkably attenuated replication and pathogenicity were generally seen. Our data further confirmed reinforcement of foreign gene expression in vitro from the V(-) version of SeV in which the accessory V gene had been knocked out. Based on these results, we discuss the utility of SeV vector in terms of both efficiency and safety.

REFERENCE COUNT: 14

REFERENCE(S):
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(7) Kato, A; J Virol 1997, V71, P7266 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:354380 CAPLUS
DOCUMENT NUMBER: 131:4238
TITLE: Construction of an alphavirus **vector** (pMP44) and its use as a DNA vaccine in immunizing against infection caused by respiratory syncytial virus or other **paramyxovirus**
INVENTOR(S): Parrington, Mark; Li, Xiaomao
PATENT ASSIGNEE(S): Connaught Laboratories Limited, Can.
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9925858	A1	19990527	WO 1998-CA1064	19981113
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9911390	A1	19990607	AU 1999-11390	19981113
EP 1034289	A1	20000913	EP 1998-954097	19981113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1997-65791 P 19971114
WO 1998-CA1064 W 19981113

AB This invention presents the construction and use of a DNA **vector** comprising a DNA sequence which is complementary to at least part of an alphavirus **RNA genome** (such as Semliki Forest virus) and having the complement of complete alphavirus DNA genome replication regions, and a second DNA sequence encoding a **paramyxovirus** (such as parainfluenza virus (PIV) or respiratory syncytial virus (RSV)) protein, such as fusion glycoprotein, G glycoprotein or hemagglutinin neuraminidase. The two DNA sequences are under the transcriptional control of a promoter, preferably a cytomegalovirus promoter, which may include intron A. The **vector** may also contain a third nucleotide sequence (such as rabbit .beta.-globin intron II) located between the promoter sequence and the alphavirus sequence to enhance the immunoprotective ability of the **paramyxovirus** protein when expressed in vivo. Finally, the DNA vector may contain a fourth nucleotide sequence (such as hepatitis delta virus ribozyme) at the 3'-end of the first nucleotide sequence to ensure proper in vivo cleavage at the 3'-end of the first nucleotide sequence. The DNA **vector** may be used to immunize a host against disease caused by infection with RSV or other **paramyxovirus**, including a human host, by administration thereto, and may be formulated as immunogenic compns. with pharmaceutically-acceptable carriers. The **vector** may also be used to produce antibodies for detection of RSV or other **paramyxovirus** infection in a sample. This invention is particularly concerned with DNA vaccines (plasmid pMP44) encoding the fusion glycoprotein of RSV in an alphavirus vector. The DNA sequence of plasmid pMP44 is claimed. The invention also described the immunization of mice with pMP44, prodn. of anti-RSV fusion glycoprotein IgG antibodies and protection of these immunized mice against live RSV challenge.

REFERENCE COUNT: 6
REFERENCE(S):
(1) Bioption AB; WO 9527044 A 1995 CAPLUS
(2) Connaught Lab; WO 9640945 A 1996 CAPLUS
(3) Connaught Lab; WO 9911808 A 1999 CAPLUS
(4) Liljestroem, P; Bio/Technology 1991, V9, P1356 CAPLUS
(5) Viagene Inc; WO 9617072 A 1996 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:189224 CAPLUS
DOCUMENT NUMBER: 130:219147
TITLE: Human respiratory syncytial virus vaccines using RNA encoding viral fusion or other glycoprotein produced from linearized vectors
INVENTOR(S): Parrington, Mark

PATENT ASSIGNEE(S): Connaught Laboratories Limited, Can.
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9911808	A1	19990311	WO 1998-CA840	19980903
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6060308	A	20000509	US 1997-923558	19970904
AU 9890569	A1	19990322	AU 1998-90569	19980903
EP 1009846	A1	20000621	EP 1998-942413	19980903
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1997-923558 A2 19970904
WO 1998-CA840 W 19980903

AB Claimed is a vector encoding a respiratory syncytial virus fusion protein which may be used to produce an RNA transcript useful as a vaccine in human. A **vector** comprising a first DNA sequence which is complementary to at least part of an alphavirus **RNA genome** and having the complement of complete alphavirus DNA genome replication regions, a second DNA sequence encoding a **paramyxovirus** protein, particularly a respiratory syncytial virus fusion (RSV F) protein or an RSV F protein fragment that generates antibodies that specifically react with RSV F protein, the first and second DNA sequences being under the transcriptional control of a promoter is described. Such **vector** may be used to produce an RNA transcript which may be used to immunize a host, including a human host, to protect the host against disease caused by **paramyxovirus**, particularly respiratory syncytial virus, by administration to the host. The RNA transcript may be formed by linearization of the vector through cleavage at a unique restriction site in a plasmid vector and then transcribing the linear mol.

REFERENCE COUNT: 6
REFERENCE(S): (1) Connaught Lab; WO 9640945 A 1996 CAPLUS
(2) Hans, L; US 5614372 A 1997 CAPLUS

(3) Liljestroem, P; BIO/TECHNOLOGY 1991, V9, P1356 CAPLUS
(4) Schlesinger, S; TRENDS IN BIOTECHNOLOGY 1993, V11(1), P18 CAPLUS
(5) Viagene Inc; WO 9617072 A 1996 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:226953 CAPLUS
DOCUMENT NUMBER: 126:208209
TITLE: Preparation of the antigenome of nonsegmented negative strand RNA viruses from cDNA with formation of infective virus and their use
INVENTOR(S): Billeter, Martin A.; Spielhofer, Pius; Kaelin, Karin; Radecke, Frank; Schneider, Henriette
PATENT ASSIGNEE(S): Schweiz. Serum- & Impfinstitut Bern, Switz.; Billeter, Martin, A.; Spielhofer, Pius; Kaelin, Karin; Radecke, Frank; Schneider, Henriette
SOURCE: PCT Int. Appl., 89 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9706270	A1	19970220	WO 1996-EP3544	19960809
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA				
EP 780475	A1	19970625	EP 1995-112559	19950809
EP 780475	B1	19990609		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
AT 181112	E	19990615	AT 1995-112559	19950809
CA 2228956	AA	19970220	CA 1996-2228956	19960809
AU 9668208	A1	19970305	AU 1996-68208	19960809
AU 721202	B2	20000629		
EP 846181	A1	19980610	EP 1996-928446	19960809
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: EP 1995-112559 A 19950809
WO 1996-EP3544 W 19960809

AB Methods for the prepn. of biol. active **genomes** of nonsegmented neg.-strand **RNA** viruses by transcription of a cDNA and the requirements for the biol. activity of the transcript are described. Virus prepd. by these methods is suitable for use in vaccines, or alternatively, as plasmids in somatic gene therapy. The invention also relates to cDNA mols. suitable as tools in this methodol. and to helper cell lines allowing the direct rescue of such viruses. Measles virus (MV) is used as a model for other representatives of the Mononegavirales, in particular the family Paramyxoviridae. For the transcript of the cDNA to be biol. active, it must be an integral multiple of six oligonucleotides long ("the rule of 6"). Methods of ensuring that the transcript obeys the rule of 6 include the use of strong terminators or the incorporation of a ribozyme into the 3'-end of the transcript to cleave the transcript at the right position.

L10 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1993:447356 CAPLUS
DOCUMENT NUMBER: 119:47356
TITLE: Improved vaccine against rhabdoviruses and paramyxoviruses
INVENTOR(S): Walker, Peter John; Prehaud, Christophe Jean
PATENT ASSIGNEE(S): Commonwealth Scientific and Industrial Research Organization, Australia
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9301833	A1	19930204	WO 1992-AU363	19920717
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
CA 2113572	AA	19930204	CA 1992-2113572	19920717
AU 9223665	A1	19930223	AU 1992-23665	19920717
EP 595970	A1	19940511	EP 1992-916278	19920717
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE				
JP 06509228	T2	19941020	JP 1992-502485	19920717
PRIORITY APPLN. INFO.:			AU 1991-7258	19910717
			WO 1992-AU363	19920717

AB A process for producing a vaccine for treatment of infections caused by rhabdoviruses and **paramyxoviruses** includes: (1) constructing a DNA corresponding to a complete genome, modified genome, or genome fragment of a rhabdovirus or **paramyxovirus**, comprising a 3'-domain, a filler domain, and a ribozyme domain;

(2) inserting the DNA obtained in 1 into the cloning site of a eukaryotic expression **vector** and transfecting a eukaryotic cell with the **vector**, simultaneously transfecting the same eukaryotic cell with **vectors** contg. cloned genes for rhabdovirus or **paramyxovirus** structural proteins, including those with similar functions to the G protein, N protein, M1 protein, and M2 protein of rabies virus; (3) obtaining from the above-transfected cell virus-like particles (VLPs) consisting of an **RNA genome** transcribed from the DNA constructed in 1 surrounded by a sheath of N protein and M1 protein to form a ribonucleoprotein complex and a lipid envelope including the G protein and an internal matrix including the M2 protein; and (4) including the VLPs obtained in 3 in a vaccine. The method of the invention was used to construct TB-2 and TB-1 DNA mols. (which contain rabies virus genome domains and filler domains and 2 and 1 ribozyme domains, resp.; TB-2 and TB-1 nucleotide sequences are included), which were incorporated into baculovirus vectors for expression in Spodoptera frugiperda cells (which also had baculovirus vectors for the rabies structural proteins). Rabies N, M1, M2, and G proteins were synthesized in all cultures infected with baculovirus expression vectors contg. the corresponding genes; however, only in cultures which were also infected with recombinant baculoviruses expressing the TB-1 and TB-2 subgenomic RNAs was there release of particles contg. the rabies proteins. The particles were also identified by electron microscopy.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 11:46:44 ON 24 MAY 2001)

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L1          137 SEA FILE=CAPLUS ABB=ON  PLU=ON  ((SHIPPING FEVER OR
                SENDAI OR BOVINE(W)(PARAINFLUENZ? OR PARA INFLUENZ?) OR
                HAEMAGGLUTIN? OR HEMAGGLUTIN? OR PARAMYXO OR PARA
                MYXO)(W)VIR? OR PARAMYXOVIR? OR PARA MYXOVIR? OR
                HVJ)(S)VECTOR
L7        23513 SEA (RNA OR RIBONUCLEIC OR RIBO NUCLEIC)(5A) GENOM?
L8           31 SEA L1 AND L7

L9           15 DUP REM L8 (16 DUPLICATES REMOVED)
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L9 ANSWER 1 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 1

ACCESSION NUMBER: 2001:187037 BIOSIS
DOCUMENT NUMBER: PREV200100187037
TITLE: Safety evaluation of hemagglutinating virus of Japan-artificial viral envelope liposomes in nonhuman primates.
AUTHOR(S): Tsuboniwa, Naoki; Morishita, Ryuichi; Hirano, Tadamichi; Fujimoto, Jiro; Furukawa, Shigenori; Kikumori, Mikito; Okuyama, Akihiko; Kaneda, Yasufumi (1)

CORPORATE SOURCE: (1) Division of Gene Therapy Science, Graduate School of Medicine, Osaka University Medical School, 2-2 Yamada-oka, Suita, Osaka, 565-0871: kaneday@gts.med.osaka-u.ac.jp Japan

SOURCE: Human Gene Therapy, (March 20, 2001) Vol. 12, No. 5, pp. 469-487. print.
ISSN: 1043-0342.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We tested, in cynomolgus monkeys, the safety and effectiveness of a hybrid liposome **vector**, **hemagglutinating virus** of Japan (**HVJ**)-artificial viral envelope (AVE) liposomes, for human therapeutic gene transfer in a series of experiments. In a repetitive intramuscular administration study, vehicle control macaques (n = 2), which were treated with **HVJ**-AVE liposome suspension, received repetitive intramuscular injections of 2 ml of test substance. Human hepatocyte growth factor (HGF) cDNA-inserted expression **vector** (pUC-SRalpha/HGF) injection animals (n = 2), which were treated with **HVJ**-AVE liposome suspension containing pUC-SRalpha/HGF, received repetitive intramuscular injection of 2 ml of test substance. General body condition, hematology, blood chemistry, and serum HGF were determined sequentially before treatment and 7, 21, 28, and 29 days after treatment. Elevations in HGF were detected in monkeys injected with pUC-SRalpha/HGF. After this observation period, macaques were killed for autopsy and histological examination. pUC-SRalpha/HGF was detected by polymerase chain reaction (PCR) analysis in the liver, spleen, and at the injection site. In single intravenous administration study, control macaques (n = 4) received a single intravenous injection of 10 ml of physiological saline. Vehicle control animals (n = 5) received a single intravenous injection of 10 ml of **HVJ**-AVE liposome suspension. DNA-treated animals (n = 7) received a single intravenous injection of 10 ml of **HVJ**-AVE liposome suspension containing plasmid DNA (pcDNA 3.1(+)). General body condition, body weight, hematology, blood chemistry, and urine composition were determined sequentially before treatment and 1, 14, 21, and 28 days after treatment. After this observation period, macaques were killed for autopsy and histological examination. pcDNA 3.1(+) was detected by PCR analysis on day 1 in lung, liver, and spleen of all monkeys, in kidney of one of two monkeys, and in heart of one of two monkeys. However, no DNA was detected in any of the tissues examined on days 14, 21, and 28. No virus **genomic RNA** was detected by reverse transcription (RT)-PCR analysis with **HVJ**-specific primers. In this series of safety evaluations, the animals tolerated the safety study with no change in body weight or general condition. No hematological changes or

alterations in blood chemistry or urine composition was detected. Moreover, no histological changes were observed. This safety evaluation study demonstrates the safety, feasibility, and therapeutic potential of the novel transfection vehicle, **HVJ** -AVE liposomes, in humans.

L9 ANSWER 2 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:520746 BIOSIS
DOCUMENT NUMBER: PREV200000520746
TITLE: RNA respiratory syncytial virus vaccines.
AUTHOR(S): Parrington, Mark (1)
CORPORATE SOURCE: (1) Bradford Canada
ASSIGNEE: Connaught Laboratories Limited, North York, Canada
PATENT INFORMATION: US 6060308 May 09, 2000
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (May 9, 2000) Vol. 1234, No. 2, pp. No pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB A **vector** comprising a first DNA sequence which is complementary to at least part of an alphavirus **RNA genome** and having the complement of complete alphavirus DNA genome replication regions, a second DNA sequence encoding a **paramyxovirus** protein, particularly a respiratory syncytial virus fusion (RSV F) protein or a RSV F protein fragment that generates antibodies that specifically react with RSV F protein, the first and second DNA sequences being under the transcriptional control of a promoter is described. Such **vector** may be used to produce an RNA transcript which may be used to immunize a host, including a human host, to protect the host against disease caused by **paramyxovirus**, particularly respiratory syncytial virus, by administration to the host.

L9 ANSWER 3 OF 15 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000405812 MEDLINE
DOCUMENT NUMBER: 20323510 PubMed ID: 10864670
TITLE: A cytoplasmic RNA **vector** derived from nontransmissible **Sendai virus** with efficient gene transfer and expression.
AUTHOR: Li H O; Zhu Y F; Asakawa M; Kuma H; Hirata T; Ueda Y; Lee Y S; Fukumura M; Iida A; Kato A; Nagai Y; Hasegawa M
CORPORATE SOURCE: DNAVEC Research Inc., Tsukuba-shi, Ibaraki 305-0856, Japan.
SOURCE: JOURNAL OF VIROLOGY, (2000 Jul) 74 (14) 6564-9.
Journal code: KCV; 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000901
Last Updated on STN: 20000901
Entered Medline: 20000822

AB We have recovered a virion from defective cDNA of **Sendai virus** (SeV) that is capable of self-replication but incapable of transmissible-virion production. This virion delivers and expresses foreign genes in infected cells, and this is the first report of a gene expression **vector** derived from a defective viral genome of the **Paramyxoviridae**. First, functional ribonucleoprotein complexes (RNPs) were recovered from SeV cloned cDNA defective in the F (envelope fusion protein) gene, in the presence of plasmids expressing nucleocapsid protein and viral RNA polymerase. Then the RNPs were transfected to the cells inducibly expressing F protein. Virion-like particles thus obtained had a titer of 0.5 x 10(8) to 1. 0 x 10(8) cell infectious units/ml and contained F-defective **RNA genome**. This defective **vector** amplified specifically in an F-expressing packaging cell line in a trypsin-dependent manner but did not spread to F-nonexpressing cells. This **vector** infected and expressed an enhanced green fluorescent protein reporter gene in various types of animal and human cells, including nondividing cells, with high efficiency. These results suggest that this **vector** has great potential for use in human gene therapy and vaccine delivery systems.

L9 ANSWER 4 OF 15 JICST-EPlus COPYRIGHT 2001 JST
ACCESSION NUMBER: 1000508178 JICST-EPlus
TITLE: Analysis of RNA polymerase activity associated with NP protein of paramyxovirus.
AUTHOR: MASAGO AKINORI
CORPORATE SOURCE: Res. Inst. for Microb. Dis., Osaka Univ.
SOURCE: Osaka Daigaku Igaku Zasshi (Medical Journal of Osaka University Japanese Edition), (2000) vol. 52, no. 3/4, pp. 125-128. Journal Code: G0933A (Fig. 3, Tbl. 1, Ref. 9)
CODEN: ODIZAK; ISSN: 0369-710X
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

AB During the life cycle of **Sendai virus**(SeV), three types of viral **RNA**(mRNA, full-length **genome** and antigenome **RNA**) are synthesized in the cytoplasm by

the viral nucleocapsid proteins(NP,P and L). Although L protein or the complex of P and L proteins have been assigned to viral RNA polymerase which utilize the NP protein-RNA complex as a template, detail mechanism of the viral RNA synthesis is still obscure. In this study, we established a cell line expressing T7 RNA polymerase stably(LLCMK2#T7) and investigated a function of NP protein of SeV. We constructed a SeV mini-genome(pHVLuciB), which directed the synthesis of (-)strand **RNA** consisting of SeV **genome** 3' leader-luciferase-SeV 5' trailer under T7 RNA polymerase promoter. When pHVLuciB was transfected to LLCMK2#T7 cells together with pGEMNP(NP protein expression **vector**), luciferase activity and (+)strand luciferase RNA were detected in the transfected cells. (+)Strand RNA synthesis required the expression of full-sized NP protein and the (-)strand template with genome 3' leader. These results suggest that SeV NP protein has intrinsic RNA polymerase activity that directs the synthesis of (+)strand RNA. (author abst.)

L9 ANSWER 5 OF 15 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-086973 [07] WPIDS
DOC. NO. CPI: C2000-024257
TITLE: Novel heat shock procedure and recombinant viruses useful for diagnostic research studies and as therapeutic or prophylactic vaccines.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): KOVACS, G R; PARKS, C L; SIDHU, M S; UDEM, S A
PATENT ASSIGNEE(S): (AMCY) AMERICAN CYANAMID CO
COUNTRY COUNT: 85
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9963064	A1	19991209	(200007)*	EN	75
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9944144	A	19991220	(200021)		
BR 9910929	A	20010220	(200114)		
EP 1090108	A1	20010411	(200121)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 9963064	A1	WO 1999-US12292	19990603
AU 9944144	A	AU 1999-44144	19990603
BR 9910929	A	BR 1999-10929	19990603
		WO 1999-US12292	19990603
EP 1090108	A1	EP 1999-927175	19990603
		WO 1999-US12292	19990603

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9944144	A	Based on	WO 9963064
BR 9910929	A	Based on	WO 9963064
EP 1090108	A1	Based on	WO 9963064

PRIORITY APPLN. INFO: US 1998-87800 19980603

AN 2000-086973 [07] WPIDS

AB WO 9963064 A UPAB: 20000209

NOVELTY - A heat shock procedure for increased recovery of recombinant Mononegavirales virus, is new.

DETAILED DESCRIPTION - A method for producing a recombinant Mononegavirales virus comprises: in at least one host cell, conducting transfection, in media, of a rescue composition which comprises: a transcription vector comprising an isolated nucleic acid molecule which comprises a polynucleotide sequence encoding a genome or anti-genome of a non-segmented, negative-sense, single stranded RNA virus of the Mononegavirales order; and at least one expression vector which comprises one or more isolated nucleic acid molecule(s) encoding the trans-acting proteins necessary for encapsidation, transcription and replication; under conditions sufficient to permit the co-expression of the vectors and the production of the recombinant virus; and heating the transfected rescue composition to an effective heat shock temperature under conditions sufficient to increase the recovery of the recombinant virus; or transferring the transfected rescue composition onto at least one layer of plaque expansion cells.

INDEPENDENT CLAIMS are also included for the following:

(1) a recombinant virus prepared by a method as above; and

(2) a composition comprising a recombinant virus prepared as above and a pharmaceutically acceptable carrier.

ACTIVITY - Anti-viral.

MECHANISM OF ACTION - Vaccine.

USE - The recombinant viruses formed by the methods are useful as tools in diagnostic research studies or as therapeutic or prophylactic vaccines. The heat shock procedure can be used to improve the efficiency of the procedure used to produce virus-like particles by packaging synthetic influenza-like CAT:**RNA** mini-**genome** in the COS-1 cells, by vaccinia-T7 polymerase

expressing cDNA clones of 10 influenza A virus-coded proteins. The method can also be used to improve efficiency of a helper independent system for the rescue of a segmented, negative strand **RNA genome** of Bunyamwera bunyavirus.

ADVANTAGE - The ability to obtain replicating virus from rescue may diminish as the polynucleotide encoding the native genome and anti-genome is increasingly modified. The methods of the invention improve the likelihood of recombinant virus rescue. An advantage of using of DNA synthesis inhibitors during a genetic rescue event is that there should be very little or no contamination of the rescued RNA virus with a modified helper virus. Heat shock temperatures above the standard temperature for performing rescue of a recombinant virus increase the recovery of the desired recombinant virus over the level of recovery of recombinant virus when rescue is performed in the absence of the increase in temperature.

Dwg.0/6

L9 ANSWER 6 OF 15 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1999-370764 [31] WPIDS
DOC. NO. CPI: C1999-109406
TITLE: Improved alphavirus **vector** containing DNA encoding a **paramyxovirus** protein.
DERWENT CLASS: B04 D16
INVENTOR(S): KLEIN, M; LI, X; PARRINGTON, M
PATENT ASSIGNEE(S): (CONN-N) CONNAUGHT LAB LTD
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9925858	A1	19990527	(199931)*	EN	61
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9911390	A	19990607	(199943)		
EP 1034289	A1	20000913	(200046)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9925858	A1	WO 1998-CA1064	19981113
AU 9911390	A	AU 1999-11390	19981113

EP 1034289	A1	EP 1998-954097	19981113
		WO 1998-CA1064	19981113

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9911390	A	Based on	WO 9925858
EP 1034289	A1	Based on	WO 9925858

PRIORITY APPLN. INFO: US 1997-65791 19971114
AN 1999-370764 [31] WPIDS
AB WO 9925858 A UPAB: 19990806

NOVELTY - An improved alphavirus **vector** containing DNA encoding an immunogenic **paramyxovirus** protein and DNA to enhance the immunoprotective ability of the **paramyxovirus** protein in vivo.

DETAILED DESCRIPTION - The vector comprises:

(1) a first DNA sequence which is complementary to at least part of an alphavirus **RNA genome** and including the complement of the complete alphavirus **RNA genome** replication regions to permit in vivo replication; and

(2) a second DNA sequence encoding a paramyxovirus protein or protein fragment that generates antibodies that specifically react with the paramyxovirus protein;

where the second DNA sequence is inserted into a region of the first DNA sequence which is non-essential for replication, the first and second DNA sequences being under transcriptional control of a promoter.

INDEPENDENT CLAIMS are also included for:

(1) a method of using a gene encoding an RSV F or G protein or fragment capable of generating antibodies which specifically react with RSV F or G protein to protect a host against disease caused by infection with RSV;

(2) producing a vaccine for protection of a host against disease caused by infection with RSV;

(3) a vaccine for administration to a host, including humans, produced as in (2); and

(4) an immunogenic composition comprising an immunoeffective amount of a vector as above.

ACTIVITY - Antiviral.

MECHANISM OF ACTION - Vaccine.

USE - The **vector** is used to immunize a host against disease caused by infection with **paramyxovirus** (claimed), especially respiratory syncytial virus. Human RSV is a major pathogen responsible for severe respiratory tract infections in infants, young children and the institutionalized elderly.

ADVANTAGE - The vector provides a protective immune response using a lower dose and less time than vectors in US8476397 and US8896500. Additionally the vectors using native RSV F produce protective immune responses in the absence of pretreatment of the animal model with cardiotoxin, a material known to increase the uptake of DNA and enhance the immune response.
Dwg.0/16

L9 ANSWER 7 OF 15 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1999-347121 [29] WPIDS
CROSS REFERENCE: 1996-251104 [25]; 1996-267677 [27]; 1996-354440 [35]; 1996-354441 [35]; 1996-354443 [35]; 1997-502401 [46]
DOC. NO. CPI: C1999-102064
TITLE: New RNA respiratory syncytial virus vaccines.
DERWENT CLASS: B04 D16
INVENTOR(S): PARRINGTON, M
PATENT ASSIGNEE(S): (CONN-N) CONNAUGHT LAB LTD
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9911808	A1	19990311	(199929)*	EN	57
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9890569	A	19990322	(199931)		
US 6060308	A	20000509	(200030)		
EP 1009846	A1	20000621	(200033)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9911808	A1	WO 1998-CA840	19980903
AU 9890569	A	AU 1998-90569	19980903
US 6060308	A	US 1997-923558	19970904
EP 1009846	A1	EP 1998-942413	19980903
		WO 1998-CA840	19980903

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9890569	A	Based on	WO 9911808
EP 1009846	A1	Based on	WO 9911808

PRIORITY APPLN. INFO: US 1997-923558 19970904
AN 1999-347121 [29] WPIDS
CR 1996-251104 [25]; 1996-267677 [27]; 1996-354440 [35]; 1996-354441 [35]; 1996-354443 [35]; 1997-502401 [46]
AB WO 9911808 A UPAB: 19990723
NOVELTY - **Vectors** containing sequences encoding a **paramyxovirus** protein, particularly Respiratory syncytial virus (RSV) F protein, which can be linearised and transcribed to RNA for in vivo administration to generate a protective immune response against the virus, are new.

DETAILED DESCRIPTION - A novel **vector** (I) comprises a first DNA sequence which is complementary to at least part of an alphavirus **RNA genome** (e.g. Semliki forest virus), and which has the complement of complete alphavirus **RNA genome** replication regions; and a second DNA sequence encoding a **paramyxovirus** protein (or fragment) that generates specific antibodies. The second DNA sequence is inserted into a region of the first sequence which is non-essential for replication. Both sequences are under the transcriptional control of a promoter, e.g. the SP6 promoter.

INDEPENDENT CLAIMS are also included for the following:

(1) a mutant DNA sequence encoding a RSV F protein or a fragment, capable of inducing antibodies that specifically react with RSV F protein, from which is about an SpeI restriction site present in the non-mutant sequence;

(2) a mutant DNA molecule lacking a SpeI site which is present in the wild type sequence, and encoding a truncated RSV F protein, and having the 1623 nucleotide sequence given in the specification or encoding the 527 amino acid sequence given in the specification;

(3) a RNA transcript of (I), which is preferably derived by linearisation of (I);

(4) an immunogenic composition for in vivo administration to a host for the generation of antibodies to paramyxoviridae protein, comprising the RNA transcript of (3) as the active component;

(5) a method of immunizing a host against disease caused by infection with paramyxovirus, which comprises administering to the host an effective amount of the RNA transcript of (3);

(6) a method of using a gene encoding a RSV F protein, comprising:

(a) isolating the gene;

(b) operatively linking the gene to a DNA sequence which is complementary to at least part of an alphavirus **RNA genome** and having the complement of complete alphavirus

RNA genome replication regions in a region of the first DNA sequence which is non-essential for replication, to form a plasmid vector, where the gene and DNA sequence are under the transcriptional control of a promoter;

(c) linearizing the plasmid vector while maintaining the gene and DNA sequence under control of the promoter;

(d) forming an RNA transcript of the linearized vector; and

(e) introducing the RNA transcript into the host; and

(7) a method of producing a vaccine for the protection of a host against disease caused by infection with RSV, comprising:

(a) isolating a first DNA sequence encoding a RSV F protein from which the transmembrane anchor and cytoplasmic tail are absent and lacking any SpeI restriction site;

(b) operatively linking the first DNA sequence to a second DNA sequence which is complementary to at least part of an alphavirus **RNA genome** and having the complement of complete alphavirus **RNA genome** replication regions in a region of the first DNA sequence which is non-essential for replication, to form a plasmid vector, wherein the both DNA sequences are under the transcriptional control of a promoter;

(c) linearizing the plasmid vector while maintaining the gene and DNA sequence under control of the promoter;

(d) forming an RNA transcript of the linearized vector; and

(e) formulating the RNA transcript as a vaccine for in vivo administration.

USE - The **vectors** and methods can be used for immunizing against disease caused by **paramyxoviridae**, including Respiratory syncytial virus (RSV) and Parainfluenza virus (PIV).

The immunogenic composition is used for the generation of protective antibodies in the host (claimed).

ADVANTAGE - The use of RNA transcripts for administration to the host enables there to total protection to challenge using a lower dose in less time than when employing present vectors. The use of RNA transcripts avoids persistence of DNA in the immunized host and potential integration. In addition, the vaccines do not cause disease enhancement (immunoprotection).

Dwg.0/4

L9 ANSWER 8 OF 15 MEDLINE DUPLICATE 3

ACCESSION NUMBER: 1999383216 MEDLINE

DOCUMENT NUMBER: 99383216 PubMed ID: 10456313

TITLE: Accommodation of foreign genes into the Sendai virus genome: sizes of inserted genes and viral replication.

AUTHOR: Sakai Y; Kiyotani K; Fukumura M; Asakawa M; Kato A; Shioda T; Yoshida T; Tanaka A; Hasegawa M; Nagai Y

CORPORATE SOURCE: Department of Viral Infection, Institute of Medical

Science, University of Tokyo, Japan.
SOURCE: FEBS LETTERS, (1999 Aug 6) 456 (2) 221-6.
Journal code: EUH; 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990913
Last Updated on STN: 19990913
Entered Medline: 19990830

AB **Sendai virus** (SeV) is an enveloped virus with a negative sense **genome RNA** of about 15.3 kb. We previously established a system to recover an infectious virus entirely from SeV cDNA and illustrated the feasibility of using SeV as a novel expression **vector**. Here, we have attempted to insert a series of foreign genes into SeV of different lengths to learn how far SeV can accommodate extra genes and how the length of inserted genes affects viral replication in cells cultured in vitro and in the natural host, mice. We show that a gene up to 3.2 kb can be inserted and efficiently expressed and that the replication speed as well as the final virus titers in cell culture are proportionally reduced as the inserted gene length increases. In vivo, such a size-dependent effect was not very clear but a remarkably attenuated replication and pathogenicity were generally seen. Our data further confirmed reinforcement of foreign gene expression in vitro from the V(-) version of SeV in which the accessory V gene had been knocked out. Based on these results, we discuss the utility of SeV **vector** in terms of both efficiency and safety.

L9 ANSWER 9 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999067521 MEDLINE
DOCUMENT NUMBER: 99067521 PubMed ID: 9850535
TITLE: New approaches to the development of virus vaccines for veterinary use.
AUTHOR: Yamanouchi K; Barrett T; Kai C
CORPORATE SOURCE: Nippon Institute for Biological Science, Tokyo, Japan.
SOURCE: REVUE SCIENTIFIQUE ET TECHNIQUE, (1998 Dec) 17 (3) 641-53. Ref: 61
Journal code: A9R; 8712301. ISSN: 0253-1933.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902

ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990209

AB The marked progress in recombinant deoxyribonucleic acid (DNA) technology during the past decade has led to the development of a variety of safe new vaccine **vectors** which are capable of efficiently expressing foreign immunogens. These have been based on a variety of virus types--poxviruses, herpesviruses and adenoviruses--and have led to the production of many new potential recombinant vaccines. Of these recombinant vaccines, the rabies vaccine, in which the rabies G protein is expressed in a vaccinia **vector**, has been widely used in the field to prevent the spread of rabies both in Europe and in the United States of America. A recombinant Newcastle disease virus vaccine, using fowlpox virus as the **vector** to express immunogenic proteins from the Newcastle disease virus, has been licensed as the first commercial recombinant vectored vaccine. Many other recombinant virus vaccines are still at the stage of laboratory or field testing. The most recent breakthrough in vaccinology has been the success with the use of naked DNA as a means of vaccination. This approach has shown great promise in mouse model systems and has now become the most active field in new vaccine development. Molecular redesigning of conventional ribonucleic acid (RNA) viruses to obtain more stable attenuated vaccines was previously possible only for positive-strand RNA viruses, such as poliovirus. However, recent advances in molecular biological techniques have enabled the rescuing of negative-strand viruses from DNA copies of their genomes. This has made it possible to engineer specific changes in the genomes of Rhabdoviridae and **Paramyxoviridae**, both of which include several viruses of veterinary importance. The authors describe the current progress in the development of **vector** vaccines, DNA vaccines and vaccines based on engineered positive- and negative-strand **RNA** virus **genomes**, with special emphasis on their application to diseases of veterinary importance.

L9 ANSWER 10 OF 15 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:268417 SCISEARCH
THE GENUINE ARTICLE: ZE342
TITLE: Large quantity production with extreme convenience of human SDF-1 alpha and SDF-1 beta by a **Sendai virus vector**
AUTHOR: Moriya C; Shioda T; Tashiro K; Nagasawa T; Ikegawa M; Ohnishi Y; Kato A; Hu H L; Xin X M; Hasan M K; Maekawa M; Takebe Y; Sakai Y; Honjo T; Nagai Y (Reprint)
CORPORATE SOURCE: UNIV TOKYO, INST MED SCI, DEPT VIRAL INFECT, MINATO KU, 4-6-1 SHIROKANEDAI, TOKYO 108, JAPAN (Reprint); UNIV TOKYO, INST MED SCI, DEPT VIRAL INFECT, MINATO

KU, TOKYO 108, JAPAN; UNIV TOKYO, INST MED SCI, DEPT INFECT DIS, TOKYO 108, JAPAN; KYOTO UNIV, CTR MOL BIOL & GENET, KYOTO 606, JAPAN; OSAKA MED CTR MATERNAL & CHILD HLTH, RES INST, DEPT IMMUNOL, OSAKA, JAPAN; KYOTO UNIV, DEPT MED CHEM, KYOTO 606, JAPAN; KYOTO UNIV, DEPT SOCIAL MED, KYOTO 606, JAPAN; KYOTO UNIV, DEPT PHARMACOL, KYOTO 606, JAPAN; NATL CTR INFECT DIS, CTR AIDS RES, DEPT MOL VIROL & EPIDEMIOL, TOKYO, JAPAN

COUNTRY OF AUTHOR: JAPAN

SOURCE: FEBS LETTERS, (20 MAR 1998) Vol. 425, No. 1, pp. 105-111.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We describe a robust expression of human stromal cell-derived factor-1 alpha (SDF-1 alpha) and SDF-1 beta, the members of CXC-chemokine family, with a novel **vector** system based upon **Sendai virus**, a non-segmented negative strand RNA virus, Recombinant SDF-1 alpha and SDF-1 beta were detected as a major protein species in culture supernatants, reached as high as 10 mu g/ml. This remarkable enrichment of the products allowed us to use even the crude supernatants as the source for biological and antiviral assays without further concentration nor purification and will thus greatly facilitate to screen their genetically engineered derivatives. (C) 1998 Federation of European Biochemical Societies.

L9 ANSWER 11 OF 15 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 96:527708 SCISEARCH
THE GENUINE ARTICLE: UW442

TITLE: A **SENDAI-VIRUS VECTOR** LEADING TO THE EFFICIENT EXPRESSION OF MUTANT M-PROTEINS INTERFERING WITH VIRUS PARTICLE BUDDING

AUTHOR: MOTTET G; MUHLEMANN A; TAPPAREL C; HOFFMANN F; ROUX L (Reprint)

CORPORATE SOURCE: CTR MED UNIV GENEVA, SCH MED, DEPT GENET & MICROBIOL, 9 AVE CHAMPEL, CH-1211 GENEVA 4, SWITZERLAND (Reprint); CTR MED UNIV GENEVA, SCH MED, DEPT GENET & MICROBIOL, CH-1211 GENEVA 4, SWITZERLAND

COUNTRY OF AUTHOR: SWITZERLAND

SOURCE: VIROLOGY, (01 JUL 1996) Vol. 221, No. 1, pp. 159-171

ISSN: 0042-6822.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 52

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A **Sendai virus** expression **vector** in the form of a transcribing copy-back defective interfering RNA was constructed and shown to efficiently express a tagged matrix protein in the only context of a **Sendai virus** infection. In an attempt to identify relevant M protein domains involved in viral assembly and budding, a series of deletion mutants were tested for their ability to bind to cellular membrane fractions. The deletion of a region spanning amino acids 105-137 significantly decreased this binding when the protein was expressed in a system driven by the T7 RNA polymerase away from any other viral proteins. Plus or minus charges were introduced in the hydrophobic portion of a predicted amphiphilic helix in this region, and M proteins with altered membrane binding properties were produced. The genes encoding these mutant M proteins were then inserted in the **Sendai virus vector** and shown to be expressed at levels similar to that of the endogenous wild-type M protein. The presence of a negative charge in the hydrophobic region of the putative amphiphilic helix prevented the incorporation of the mutant protein into virus particles and appeared to decrease the efficiency of virus particle budding. In contrast, the introduction of a positive charge appeared to increase the M mutant uptake into virions. The use a **Sendai virus vector** has therefore been shown instrumental in the identification of mutant M proteins interfering with the viral assembly-budding process. (C) 1996 Academic Press, Inc.

L9 ANSWER 12 OF 15 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 94201756 MEDLINE
DOCUMENT NUMBER: 94201756 PubMed ID: 8151297
TITLE: The nucleoprotein of Marburg virus is phosphorylated.
AUTHOR: Becker S; Huppertz S; Klenk H D; Feldmann H
CORPORATE SOURCE: Institut fur Virologie, Marburg, Germany.
SOURCE: JOURNAL OF GENERAL VIROLOGY, (1994 Apr) 75 (Pt 4) 809-18.
Journal code: I9B; 0077340. ISSN: 0022-1317.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940523

Last Updated on STN: 19940523
Entered Medline: 19940509

AB The nucleoprotein (NP) of Marburg virus (MBG), a filovirus, is encoded by the gene closest to the 3' end of the non-segmented negative-strand **RNA genome**. Sequence comparison has indicated that NP is the functional equivalent to the nucleoproteins of **paramyxoviruses** and rhabdoviruses. Expression of recombinant NP in two eukaryotic systems using vaccinia virus and baculovirus (**vectors** pSC11 and pAcYMB1, respectively) and analysis of MBG-specific proteins have demonstrated that the NP of MBG is phosphorylated. The NP appeared in two forms differing in M(r) by about 2K (94K and 92K respectively). Dephosphorylation clearly demonstrated that the 94K form is phosphorylated whereas the 92K form is unphosphorylated. In virion particles NP was exclusively present in the phosphorylated form. These findings suggest that only the phosphorylated NP can form nucleocapsid complexes and interact with the **genomic RNA**.

L9 ANSWER 13 OF 15 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1993-058528 [07] WPIDS
DOC. NO. CPI: C1993-026124
TITLE: Vaccine prepn against rhabdo- and para myxoviruses - includes virus-like particle comprising **RNA genome** with 3' and filler domain and protein sheath of rabies M protein or similar protein.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): PREHAUD, C J; WALKER, P J
PATENT ASSIGNEE(S): (CSIR) COMMONWEALTH SCI & IND RES ORG
COUNTRY COUNT: 20
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9301833	A1	19930204	(199307)*	EN	43
RW: AT BE CH DE DK ES FR GB GR IT LU MC NL SE					
W: AU CA JP US					
AU 9223665	A	19930223	(199324)		
NZ 243611	A	19931223	(199403)		
EP 595970	A1	19940511	(199419)	EN	
R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE					
JP 06509228	W	19941020	(199501)		
EP 595970	A4	19950531	(199615)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 9301833	A1	WO 1992-AU363	19920717
AU 9223665	A	AU 1992-23665	19920717
NZ 243611	A	NZ 1992-243611	19920717
EP 595970	A1	EP 1992-916278	19920717
		WO 1992-AU363	19920717
JP 06509228	W	WO 1992-AU363	19920717
		JP 1993-502485	19920717
EP 595970	A4	EP 1992-916278	

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9223665	A	Based on	WO 9301833
EP 595970	A1	Based on	WO 9301833
JP 06509228	W	Based on	WO 9301833

PRIORITY APPLN. INFO: AU 1991-7258 19910717
AN 1993-058528 [07] WPIDS
AB WO 9301833 A UPAB: 19931119
Prodn. of a vaccine for treatment of infections cuased by rhabdoviruses (RVs) and **paramyxoviruses** (PVs) includes (i) constructing DNA mol. corresp. to completed or modified genome or fragment of the virus comprising 3' domain, a filler domain and ribozyme domain; (ii) inserting the DNA into a cloning site of a eukaryotic expression **vector** and simultaneously transfecting eukaryotic cell with this **vector** and **vectors** contg. cloned genes of the virus structural proteins, including those with similar functions to the G, N, M1 and M2 proteins of rabies virus; (iii) obtaining from the cell virus-like particles (VLPs) consisting of **RNA genome** transcribed from the DNA mol. in (i) surrounded by sheath of N and M1 proteins to form tribonucleoprotein complex, with lipid envelope including the G protein and an internal matrix of M2; and (iv) including the VLPs in vaccine.

Also new are: (a) the vaccine prepared; (b) a VLP; and (c) a DNA construct including 3', ribozyme and filler domains.

USE/ADVANTAGE - The vaccine against RVs and PVs is effective and completely non-infectious

Dwg. 0/12

L9 ANSWER 14 OF 15 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 93079897 MEDLINE
DOCUMENT NUMBER: 93079897 PubMed ID: 1333129
TITLE: Sequence characterization and expression of the matrix protein gene of human parainfluenza virus type 1.

AUTHOR: Power U F; Ryan K W; Portner A
CORPORATE SOURCE: Department of Virology and Molecular Biology, St. Jude Children's Research Hospital, Memphis, Tennessee 38101.
CONTRACT NUMBER: AI 11949 (NIAID)
AI 31596 (NIAID)
CA21765 (NCI)
SOURCE: VIROLOGY, (1992 Dec) 191 (2) 947-52.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M80818
ENTRY MONTH: 199212
ENTRY DATE: Entered STN: 19930129
Last Updated on STN: 19970203
Entered Medline: 19921230

AB The nucleotide sequence of the M gene of human parainfluenza virus type 1 (hPIV1) was determined from **genomic RNA** and cDNA copies of the entire gene. The M gene contained 1173 nucleotides. It had one large open reading frame capable of encoding a protein of 348 amino acids (M(r) = 38,404). The predicted amino acid sequence of the hPIV1 M protein is highly basic (+20 at neutral pH). A pGEM-1 expression **vector** containing the M gene was used for cell-free transcription and translation. The resultant protein was confirmed to be M by electrophoretic mobility and immunoprecipitation. Among other **paramyxoviridae** the hPIV1 M amino acid sequence was most closely related to the **Sendai virus** M sequence (87% identity). The pattern of M gene relatedness observed from the alignment of 16 **paramyxoviridae** M protein amino acid sequences was not predicted by the viruses' taxonomic classification.

L9 ANSWER 15 OF 15 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 83221565 MEDLINE
DOCUMENT NUMBER: 83221565 PubMed ID: 6190173
TITLE: cDNA cloning and transcriptional mapping of nine polyadenylylated **RNAs** encoded by the **genome** of human respiratory syncytial virus.
AUTHOR: Collins P L; Wertz G W
CONTRACT NUMBER: A112464 (NCI)
A115134
CA09156
+
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1983 Jun) 80 (11) 3208-12.

Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198307
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19830708

AB We have isolated cDNA clones representing nine unique poly(A)+ **RNAs** transcribed from the **genome** of human respiratory syncytial virus, a **paramyxovirus**. A cDNA library was constructed by using poly(A)+ RNA from virus-infected cells as template and the Escherichia coli plasmid pBR322 as **vector**. Viral cDNA clones were identified by hybridization with cDNA probes prepared from viral **genomic RNA** . The viral clones were grouped into nine different families by hybridization with individual size-selected reverse transcripts representing the major classes of poly(A)+ RNA from virus-infected cells. The largest clone from each family was selected for analysis. These nine clones, molecular sizes ranging from 520 to 2,600 base pairs, were shown to be unrelated on the basis of reciprocal hybridization using dot-blots. These cDNA clones were then used as hybridization probes to analyze intracellular viral RNAs that had been separated by gel electrophoresis and transferred to diazobenzyloxymethyl-paper. All nine clones hybridized with intracellular viral **genomic RNA**, confirmation of virus specificity. Nine unique intracellular viral poly(A)+ RNAs were identified [molecular sizes ranging from 720 to 7,500 nucleotides, including poly(A)]. Comparison of the sizes of these major RNAs and the cDNA clones indicated that a number of the clones represented nearly complete copies of the corresponding RNAs. Several other intracellular viral poly(A)+ RNAs appeared to be polycistronic by the criteria of molecular weights and homologies to various combinations of cDNA clones. The sizes and sequence contents of these polycistronic RNAs were used to prepare a transcriptional map whose significance is discussed.

(FILE 'CAPLUS' ENTERED AT 11:54:29 ON 24 MAY 2001)
L11 259 SEA FILE=CAPLUS ABB=ON PLU=ON SEV OR (HPIV OR PV OR PIV)(S)PARAMYXOVIR?
L12 11 SEA FILE=CAPLUS ABB=ON PLU=ON L11 AND ((RNA OR RIBONUCLEIC OR RIBO NUCLEIC)(5A)GENOM?)

=> s l12 not l10
L13 7 L12 NOT L10

L13 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1998:258483 CAPLUS
DOCUMENT NUMBER: 129:38615
TITLE: The short Sendai virus leader region controls induction of programmed cell death
AUTHOR(S): Garcin, Dominique; Taylor, Geraldine; Tanebayashi, Kiyoshi; Compans, Richard; Kolakofsky, Daniel
CORPORATE SOURCE: Department of Genetics and Microbiology, University of Geneva School of Medicine, CMU, Geneva, CH1211, Switz.
SOURCE: Virology (1998), 243(2), 340-353
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The replication of nonsegmented minus-strand **RNA genomes**, like that of Sendai paramyxovirus (**SeV**), are controlled by the short leader regions present at each end of the linear genomes and antigenomes; the left and right promoters (PL and PR), resp. Wild-type **SeV** is highly cytopathic in cell culture, because it induces programmed cell death (PCD). We have found that a recombinant **SeV** (rSeVGP42), in which the first 42 nt of le+ sequences at PL were replaced with the equiv. sequences of PR, and which produces infectious virus in amts. comparable to wild type, does not kill cells. Further, the increasing replacement of the terminal le+ sequences at PL with le- sequences led to a decreasing fraction of infected cells being apoptotic. This property (PCD-), moreover, is dominant in cells co-infected with SeVwt and rSeVGP42, and the mutant virus therefore appears to have gained a function which prevents PCD induced by SeVwt. Even though this virus has not been selected for naturally, it excludes SeVwt during co-infections of cultured cells or embryonated chicken eggs. The noncytopathic nature of cells infected or co-infected with rSeVGP42 leads automatically to stable, persistent infections. The mutation in rSeVGP42 is not in the protein coding regions of the viral genome, but in the 55-nt-long leader region which controls antigenome synthesis from genome templates. The **SeV** leader regions, which are expressed as short RNAs, thus appear to control the induction of PCD.

L13 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1991:442830 CAPLUS
DOCUMENT NUMBER: 115:42830
TITLE: Molecular cloning and sequence analysis of human parainfluenza type 2 virus mRNA encoding the fusion glycoprotein
AUTHOR(S): Varsanyi, Tamas M.; Kovamees, Jan; Norrby, Erling

CORPORATE SOURCE: Sch. Med., Karolinska Inst., Stockholm, S-105 21, Swed.
SOURCE: J. Gen. Virol. (1991), 72(1), 89-95
CODEN: JGVIAY; ISSN: 0022-1317
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A library of cDNA clones from mRNA of human parainfluenza type 2 virus (PIV2) was constructed and the nucleotide sequence of the fusion (F) glycoprotein gene detd. The F gene boundaries were obtained by primer extension sequencing on F mRNA and on viral **genomic RNA**. The mRNA coding for the F glycoprotein is composed of 1918 nucleotides. It contains a single large open reading frame that encodes a protein of 551 amino acids with an Mr of 59,586. The predicted **PIV** 2 F protein contains the cleavage-activation site (amino acids 101 to 106) including 2 Arg and two Lys residues, where the protein is cleaved by host protease into F1 and F2 subunits by analogy with other **paramyxoviruses**. Three hydrophobic domains are recognized, the signal peptide (amino acids 1 to 21), that is cleaved off in the mature protein, the fusion peptide (amino acids 107 to 132) at the cleavage-generated N terminus of subunit F1 and the membrane anchorage region (amino acids 486 to 513) near the C terminus of the protein. The predicted F protein has six potential glycosylation sites and 10 of the 12 Cys residues present have conserved positions as compared with those of other paramyxovirus F proteins. Primer extension sequencing on viral RNA gave the 3' end sequence as UAAAUUCU6 followed by the 5' end of the hemagglutinin-neuraminidase (HN) gene thus establishing the order of genes coding for the viral glycoproteins of PIV2 as 5'-F-HN-3'.

L13 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1991:200562 CAPLUS
DOCUMENT NUMBER: 114:200562
TITLE: Sequence analyses of the 3' genome end and NP gene of human parainfluenza type 2 virus: sequence variation of the gene-starting signal and the conserved 3' end
AUTHOR(S): Yuasa, Tetsuya; Bando, Hisanori; Kawano, Mitsuo; Tsurudome, Masato; Nishio, Machiko; Kondo, Kunio; Komada, Hiroshi; Ito, Yasuhiko
CORPORATE SOURCE: Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE: Virology (1990), 179(2), 777-84
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The nucleotide sequences of cDNAs against nucleocapsid protein (NP) mRNA and the **genomic RNA** of human parainfluenza type 2 virus (PIV-2) were detd. The 3'-terminal region of

genomic RNA was compared among PIV-2, mumps virus (MuV), Newcastle disease virus (NDV), measles virus (MV), PIV-3, bovine parainfluenza type 3 virus (BPIV-3), Sendai virus (SV), and vesicular stomatitis virus (VSV), and an extensive sequence homol. was obsd. between PIV-2 and MuV. Although no significant sequence relatedness was obsd. between **PIV**-2 and other viruses, the terminal 4 nucleotides were identical in the viruses compared, implying a specific role of these nucleotides in the replication of **paramyxoviruses**. A primer extension anal. elucidated the major NP mRNA initiation site as the sequence UCUAAGCC, which showed a moderate homol. with the gene-starting consensus sequences of other paramyxoviruses. On the other hand, the NP mRNA was terminated at the nucleotide stretch AAAUUCUUUUU, and this sequence was conserved in all the PIV-2 genes, indicating that the oligonucleotides will form a part of the gene attenuation signal of PIV-2. Comparisons of the NP protein sequence indicated a possible subgrouping of the **paramyxoviruses** into 2 groups, one of which is a group including **PIV**-2, **PIV**-4, MuV, and NDV, and another is a group including **PIV**-3, BPIV-3, and SV. This result supports an idea from previous studies using polyclonal and monoclonal antibodies. Furthermore, data indicated that the PIV-2 NP protein sequence was more closely related to MV and CDV than to other parainfluenza viruses, PIV-3 and SV.

L13 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER:	1991:1332 CAPLUS
DOCUMENT NUMBER:	114:1332
TITLE:	Sequence of the fusion protein gene of human parainfluenza type 2 virus and its 3' intergenic region: lack of small hydrophobic (SH) gene
AUTHOR(S):	Kawano, Mitsuo; Bando, Hisanori; Ohgimoto, Shinji; Kondo, Kunio; Tsurudome, Masato; Nishio, Machiko; Ito, Yasuhiko
CORPORATE SOURCE:	Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE:	Virology (1990), 178(1), 289-92 CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Some cDNA clones representing the fusion (F) gene of human parainfluenza virus type 2 (PIV-2) were isolated from cDNA libraries constructed from virus-specific mRNA and **genomic RNA**, and the complete nucleotide sequence of the F gene was detd. The F gene is 1854 nucleotides long and encodes 551 amino acids. The cleavage site for activation of the precursor F0 proteins is Thr-Arg-Gln-Lys-Arg. The F gene of PIV-2 is most closely related to those of simian virus 5 (SV5) and mumps virus (MuV). Although the HN glycoprotein of PIV-2 shows no relatedness to the HA glycoprotein of measles virus (MV), a distinct homol. is

found in the F proteins of PIV-2 and MV. As concerns F proteins, **paramyxoviruses** can be divided into two subgroups that is **PIV**-2, SV5, and MuV belong to one group, and **HPIV** -1, SV, and **PIV**-3 belong to the other group. Newcastle disease virus (NDV) and MV are intermediate. Coding regions for small hydrophobic (SH) proteins have been found between the HN and F genes of SV5 and MuV, which are the viruses most closely related to PIV-2. However, such a gene could not be detected in two different strains of PIV-2.

L13 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1990:625287 CAPLUS
DOCUMENT NUMBER: 113:225287
TITLE: Sequence analysis of the phosphoprotein (P) genes of human parainfluenza type 4A and 4B viruses and RNA editing at transcript of the P genes: the number of G residues added is imprecise
AUTHOR(S): Kondo, Kunio; Bando, Hisanori; Tsurudome, Masato; Kawano, Mitsuo; Nishio, Machiko; Ito, Yasuhiko
CORPORATE SOURCE: Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE: Virology (1990), 178(1), 321-6
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors cloned and sequenced the cDNAs against **genomic RNAs** and mRNAs for phosphoproteins (Ps) of human parainfluenza virus types 4A (PIV-4A) and 4B (PIV-4B). The PIV-4A and -4B P genes were 1535 nucleotides including poly(A) tract and were found to have 2 small open reading frames, neither of which was apparently large enough to encode the P protein. A cluster of G residues was found in **genomic RNA**, and the no. of G residues was 6 in both PIV-4A and -4B. However, the no. of G residues at the corresponding site in the mRNAs to the **genomic RNA** was not const. Three different mRNA cDNA clones were obtained; the first type of mRNA encodes a larger (P) protein of 399 amino acids, the second type encodes V protein of 229 or 230 amino acids, and the third type encodes the smallest protein (156 amino acids). Comparisons on the nucleotide and the amino acid sequences of P and V proteins between these 2 subtypes revealed extensive homologies. However, these homol. degrees are lower than that of NP protein. The C-terminal regions of the P and V proteins of **PIV**-4s could be aligned with all other **paramyxoviruses**, **PIV**-2, mumps virus (MuV), simian virus 5 (SV 5), Newcastle disease virus (NDV), measles virus (MV), canine distemper virus (CDV), Sendai virus (SV), and **PIV** -3. On the other hand, the P-V common (N-terminal) regions showed

no homol. with MV, CDV, SV, and PIV-3. Seven phylogenetic trees of Paramyxoviruses were constructed from the entire and partial regions of P and V proteins.

L13 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1990:605627 CAPLUS
DOCUMENT NUMBER: 113:205627
TITLE: Sequence analysis of P gene of human parainfluenza type 2 virus: P and cysteine-rich proteins are translated by two mRNAs that differ by two nontemplated G residues
AUTHOR(S): Ohgimoto, Shinji; Bando, Hisanori; Kawano, Mitsuo; Okamoto, Kousuke; Kondo, Kunio; Tsurudome, Masato; Nishio, Machiko; Ito, Yasuhiko
CORPORATE SOURCE: Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE: Virology (1990), 177(1), 116-23
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The cDNAs against **genomic RNA** and mRNA for phosphoprotein (P) of human parainfluenza type 2 virus (PIV-2) were cloned and sequenced. The cDNA clone from **genomic RNA** was 1439 nucleotides in length excluding poly(A) and had 2 small open reading frames encoding proteins of 233 and 249 amino acids. Two different mRNA cDNA clones were obtained; i.e., one mRNA contained a smaller reading frame coding 225 amino acids, V protein, and the other mRNA contained a larger reading frame coding 395 amino acids, P protein. Both mRNAs had G clusters in their coding frame. The former mRNA contained 7 G residues , and 2 extra G residues were inserted in the latter mRNA. Ten cDNA clones from the **genomic RNA** were identical and were composed to 7 G residues, indicating that genomes analyzed here were a homogeneous population. Therefore, V protein is encoded by faithfully copied mRNA and P protein is translated from mRNA in which 2 addnl. G residues are nontemplately inserted immediately after 7 genomically encoded G residues. The V and P proteins are amino coterminal proteins and have different C termini. The C terminus of V protein is cysteine-rich and bears some resemblance to metal-binding protein of the zinc finger-type motif. The P protein sequence of PIV-2 showed high homologies with SV 5 (40.4%) and mumps virus (35.5%), and a moderate homol. with Newcastle disease virus (20.6%). On the other hand, very little homol. was found between **PIV**-2 and other **paramyxoviruses** including Sendai virus, **PIV**-3, and measles virus. The cysteine-rich region in V protein was found to be highly conserved in **PIV**-2, SV 5, and measles virus, suggesting that V protein of **paramyxoviruses** plays important roles in transcription and/or replication. The predicted

cysteine-rich V protein was detected in virus-infected cells using antiserum directed against an oligopeptide specific for the predicted V polypeptide.

L13 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1990:453269 CAPLUS
DOCUMENT NUMBER: 113:53269
TITLE: Molecular cloning and sequence analysis of human parainfluenza type 4A virus HN gene: its irregularities on structure and activities
AUTHOR(S): Bando, Hisanori; Kondo, Kunio; Kawano, Mitsuo; Komada, Hiroshi; Tsurudome, Masato; Nishio, Machiko; Ito, Yasuhiko
CORPORATE SOURCE: Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE: Virology (1990), 175(1), 307-12
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The cDNA of human parainfluenza type 4A virus (PIV-4A) NH gene was cloned by reverse-transcription of virus-specific mRNAs and **genomic RNA**, and the complete nucleotide sequence of the NH gene was detd. The predicted HN protein sequence of **PIV**-4A showed significant relatedness with those of other **paramyxoviruses**, SV5, NDV, MuV, **PIV**-3, BPIV-3, indicating a common ancestor. The homologies between the viruses suggested that PIV-4A is more closely related to NDV, SV5, and MuV than to the parainfluenza viruses, PIV-3, bovine parainfluenza type 3 virus (BPIV-3), and Sendai virus (SV). Sixty amino acids were commonly conserved among the viruses, other than PIV-4A. Two of these amino acids were substituted in PIV-4A HN and are predicted to be located near the active site of the neuraminidase. The anal. of neuraminidase of PIV-4 revealed that the activity is hardly detectable, suggesting the significant effect of the substituted amino acid sites on neuraminidase activity.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 11:57:26 ON 24 MAY 2001)
L14 39 S L12
L15 26 S L14 NOT L8
L16 10 DUP REM L15 (16 DUPLICATES REMOVED)

L16 ANSWER 1 OF 10 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2001-257914 [26] WPIDS
DOC. NO. CPI: C2001-077742
TITLE: Treating tumor by administering replication-competent Paramyxoviridae virus comprising nucleic acid encoding cytokine or heterologous polypeptide whose detection in

biological fluid indicates reduction in tumor size.

DERWENT CLASS: B04 C06 D16
INVENTOR(S): CATTANEO, R; MURPHY, A L; PENG, K; RUSSELL, J; SCHNEIDER, U
PATENT ASSIGNEE(S): (MAYO-N) MAYO FOUND MEDICAL EDUCATION & RES
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001020989	A1	20010329	(200126)*	EN	102

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001020989	A1	WO 2000-US26116	20000922

PRIORITY APPLN. INFO: US 1999-155873 19990922
AN 2001-257914 [26] WPIDS
AB WO 200120989 A UPAB: 20010515
NOVELTY - Treating (I) tumor in a patient, comprises administering a replication-competent **Paramyxoviridae** virus (**PV**) comprising:

(a) a nucleic acid (NA) sequence encoding a heterologous polypeptide (PP), where detection of PP in biological fluid is indicative of PV growth in patient and reduction in tumor size and/or;

(b) a recombinant F, H or M protein of PV that increases cell-cell fusion rate of virus with tumor cells.

DETAILED DESCRIPTION - (I) Comprises administering PV comprising two or more of:

(i) a NA sequence encoding a heterologous PP whose detection in biological fluid is indicative of PV growth and reduction in tumor size;

(ii) a recombinant F, H or M protein of PV that increases cell-cell fusion rate of virus with tumor cells;

(iii) a NA sequence encoding a cytokine; and

(iv) a PV specific for tumor cells.

INDEPENDENT CLAIMS are also included for the following:

(1) increasing cell-cell fusion rate of PV on tumor cells, by contacting tumors cells with a replication-competent PV comprising one or more of recombinant F, H or M protein of the PV that increases cell-cell fusion rate of the virus with the cells;

(2) monitoring reduction in tumor size in a patient, by administering a replication-competent PV comprising a nucleic acid sequence encoding a heterologous PP, where detection of PP in biological fluid is indicative of PV growth in the patient and reduction in tumor size;

(3) producing (II) a recombinant PV, by:

(a) transfecting an eukaryotic cell line stably expressing T7 **RNA** polymerase with an infectious PV **genomic** cDNA under the control of T7 promoter;

(b) infecting the transfected cells with a helper virus expressing a selectable trait and N, P and L proteins of PV;

(c) contacting the infected, transfected cells with cells that permit PV infection and replication, under conditions permitting the infection and replication;

(d) selecting syncytia formed on the cells that permit PV infection and replication;

(e) screening for and isolating PV lacking helper viral genetic material based upon the presence or absence of selectable trait of the helper virus; and

(f) expanding PV lacking helper genetic material to produce recombinant PV; and

(4) a kit for treatment of a patient having a tumor, comprising a replication-competent PV.

ACTIVITY - Anti-tumor.

No supporting data is given.

MECHANISM OF ACTION - Gene therapy.

USE - The method is useful for treating a patient having a tumor in order to reduce tumor size.

Dwg.0/19

L16 ANSWER 2 OF 10 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 2000:406992 SCISEARCH
THE GENUINE ARTICLE: 317LP
TITLE: Structure-function analysis of the Sendai virus F and HN cytoplasmic domain: Different role for the two proteins in the production of virus particle
AUTHOR: FouillotCoriou N; Roux L (Reprint)
CORPORATE SOURCE: UNIV GENEVA, SCH MED, DEPT GENET & MICROBIOL, CMU, 1 RUE MICHEL SERVET, CH-1211 GENEVA 4, SWITZERLAND (Reprint); UNIV GENEVA, SCH MED, DEPT GENET & MICROBIOL, CMU, CH-1211 GENEVA 4, SWITZERLAND
COUNTRY OF AUTHOR: SWITZERLAND
SOURCE: VIROLOGY, (10 MAY 2000) Vol. 270, No. 2, pp. 464-475

Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0042-6822.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 45

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The role of the cytoplasmic domain (cytd) of the Sendai virus HN and F glycoproteins in the process of virus assembly and budding are evaluated. Recombinant Sendai virus (rSeV) mutants are generated carrying modifications In the cytd of each of the glycoprotein separately. The modifications include increasing truncations and/or amino acid sequence substitutions. Following steady-state (35)[S]methionine/cysteine labeling of the infected cells, the virus particle production is estimated. The radioactive virions in the cell supernatants are measured relative to the extent of the infection, assessed by the Intracellular N protein signal. For both the F and HN cytd truncation mutants, the largest cytd deletions lead to a 20- to 50-fold reduction in virion production. This reduction cannot be explained by a reduction of the cell surface expression of the glycoproteins. For the F protein mutants, the virions produced in reduced amount always exhibit a normal F protein composition. It is then concluded that a threshold level of F is required for **SeV** assembly and budding. The rate or the efficiency with which this threshold is reached up appears to depend on the nature of the F cytd. A minimal cytd length is required as well as a specific sequence. The analysis of HN protein mutants brings to light an apparent paradox. The larger cytd truncations result In significant reduction of virion production. On the other hand, a normal virion production can take place with an underrepresentation of or, even, an undetectable HN in the particles. The HN uptake in virion is confirmed to depend on the previously proposed cytd SYWST signal (T. Takimoto, T Bousse, E. C. Coronel, R. A Scroggs, and A. Portner. 1998. J. Virol. 72, 9747-9754). (C) 2000 Academic Press.

L16 ANSWER 3 OF 10 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1998229453 MEDLINE
DOCUMENT NUMBER: 98229453 PubMed ID: 9568033
TITLE: The short Sendai virus leader region controls induction of programmed cell death.
AUTHOR: Garcin D; Taylor G; Tanebayashi K; Compans R; Kolakofsky D
CORPORATE SOURCE: Department of Genetics and Microbiology, University of Geneva School of Medicine, CMU, Switzerland.
SOURCE: VIROLOGY, (1998 Apr 10) 243 (2) 340-53.
Journal code: XEA; 0110674. ISSN: 0042-6822.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980609
Last Updated on STN: 19990129
Entered Medline: 19980527

AB The replication of nonsegmented minus-strand **RNA genomes**, like that of Sendai paramyxovirus (**SeV**), are controlled by the short leader regions present at each end of the linear genomes and antigenomes; the left and right promoters (PL and PR), respectively. Wild-type **SeV** is highly cytopathic in cell culture, because it induces programmed cell death (PCD). We have found that a recombinant **SeV** (rSeVGP42), in which the first 42 nt of le+ sequences at PL were replaced with the equivalent sequences of PR, and which produces infectious virus in amounts comparable to wild type, does not kill cells. Further, the increasing replacement of the terminal le+ sequences at PL with le- sequences led to a decreasing fraction of infected cells being apoptotic. This property (PCD-), moreover, is dominant in cells co-infected with SeVwt and rSeVGP42, and the mutant virus therefore appears to have gained a function which prevents PCD induced by SeVwt. Even though this virus has not been selected for naturally, it excludes SeVwt during co-infections of cultured cells or embryonated chicken eggs. The noncytopathic nature of cells infected or co-infected with rSeVGP42 leads automatically to stable, persistent infections. The mutation in rSeVGP42 is not in the protein coding regions of the viral genome, but in the 55-nt-long leader region which controls antigenome synthesis from genome templates. The **SeV** leader regions, which are expressed as short RNAs, thus appear to control the induction of PCD.

L16 ANSWER 4 OF 10 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 96:59250 SCISEARCH
THE GENUINE ARTICLE: TP210
TITLE: A HIGHLY RECOMBINOGENIC SYSTEM FOR THE RECOVERY OF INFECTIOUS SENDAI-PARAMYXOVIRUS FROM CDNA - GENERATION OF A NOVEL COPY-BACK NONDEFECTIVE INTERFERING VIRUS
AUTHOR: GARCIN D (Reprint); PELET T; CALAIN P; ROUX L; CURRAN J; KOLAKOFSKY D
CORPORATE SOURCE: UNIV GENEVA, SCH MED, DEPT GENET & MICROBIOL, CMU, 9 AVE CHAMPEL, CH-1211 GENEVA, SWITZERLAND (Reprint)
COUNTRY OF AUTHOR: SWITZERLAND
SOURCE: EMBO JOURNAL, (15 DEC 1995) Vol. 14, No. 24, pp. 6087-6094.
ISSN: 0261-4189.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 32
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We have recovered infectious Sendai virus (**SeV**) from full-length cDNA (FL-3) by transfecting this cDNA and pGEM plasmids expressing the nucleocapsid protein (NP), phosphoprotein and large proteins into cells infected with a vaccinia virus which expresses T7 RNA polymerase, These cells were then injected into chicken eggs, in which **SeV** grows to very high titers, FL-3 was marked with a BglII site in the leader region and an NsiI site (ATGCAT) in the 5' nontranslated region of the NP gene, creating a new, out-of-frame, 5' proximal AUG, All the virus stocks generated eventually removed this impediment to NP expression, by either point mutation or recombination between FL-3 and pGEM-NP, The recovery system was found to be highly recombinogenic, Even in the absence of selective pressure, one in 20 of the recombinant **SeV** generated had exchanged the NP gene of FL-3 with that of pGEM-NP. When a fifth plasmid containing a new genomic 3' end without the presumably deleterious BglII site was included as another target for recombination, the new genomic 3' end was found in the recombinant **SeV** in 12 out of 12 recoveries, Using this approach, a novel copy-back nondefective virus was generated which interferes with wild-type virus replication.

L16 ANSWER 5 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 91252221 MEDLINE
DOCUMENT NUMBER: 91252221 PubMed ID: 1645865
TITLE: Characterizations of the human parainfluenza type 2 virus gene encoding the L protein and the intergenic sequences.
AUTHOR: Kawano M; Okamoto K; Bando H; Kondo K; Tsurudome M; Komada H; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Japan.
SOURCE: NUCLEIC ACIDS RESEARCH, (1991 May 25) 19 (10) 2739-46.
Journal code: O8L; 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-X55368; GENBANK-X55369; GENBANK-X55370; GENBANK-X57505; GENBANK-X57506; GENBANK-X57507; GENBANK-X57508; GENBANK-X57509; GENBANK-X57510; GENBANK-X57559
ENTRY MONTH: 199107

ENTRY DATE: Entered STN: 19910728
Last Updated on STN: 19970203
Entered Medline: 19910711

AB We cloned and determined the nucleotide sequences of cDNAs against **genomic RNA** encoding the L protein of human parainfluenza type 2 virus (**PIV**-2). The L gene is 6904 nucleotides long including the intergenic region at the HN-L junction and putative negative strand leader RNA, almost all of which is complementary to the positive strand leader RNA of **PIV**-2. The deduced L protein contains 2262 amino acids with a calculated molecular weight of 256,366. The L protein of **PIV**-2 shows 39.9, 28.9, 27.8 and 28.3% homologies with Newcastle disease virus (NDV), Sendai virus (SV), parainfluenza type 3 virus (**PIV**-3) and measles virus (MV), respectively. Although sequence data on other components of transcriptive complex, NP and P, suggested a closer relationship between **PIV**-2 and MV, as concerns the L protein, MV is closely related to another group as SV and **PIV**-3. From analysis of the alignment of the five l proteins, six blocks composed of conserved amino acids were found in the L proteins. The L protein of **PIV**-2 was detected in purified virions and virus-infected cells using antiserum directed against an oligopeptide corresponding to the amino terminal region. Primer extension analyses showed that the intergenic regions at the NP-P, P-M, M-F, F-HN and HN-L junctions are 4, 45, 28, 8 and 42 nucleotides long, respectively, indicating that the intergenic regions exhibit no conservation of length and sequence. Furthermore, the starting and ending sequences of **paramyxoviruses** were summarized.

L16 ANSWER 6 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 91049444 MEDLINE
DOCUMENT NUMBER: 91049444 PubMed ID: 2173261
TITLE: Sequence analyses of the 3' genome end and NP gene of human parainfluenza type 2 virus: sequence variation of the gene-starting signal and the conserved 3' end.
AUTHOR: Yuasa T; Bando H; Kawano M; Tsurudome M; Nishio M; Kondo K; Komada H; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Japan.
SOURCE: VIROLOGY, (1990 Dec) 179 (2) 777-84.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M55320
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 19910208

Last Updated on STN: 19980206
Entered Medline: 19901221

AB We cloned and determined the nucleotide sequences of cDNAs against nucleocapsid protein (NP) mRNA and the **genomic RNA** of human parainfluenza type 2 virus (**PIV**-2). The 3' terminal region of **genomic RNA** was compared among **PIV**-2, mumps virus (MuV), Newcastle disease virus (NDV), measles virus (MV), **PIV**-3, bovine parainfluenza type 3 virus (BPIV-3), Sendai virus (SV), and vesicular stomatitis virus (VSV), and an extensive sequence homology was observed between **PIV**-2 and MuV. Although no significant sequence relatedness was observed between **PIV**-2 and other viruses, the terminal four nucleotides were identical in the viruses compared, implying a specific role of these nucleotides on the replication of **paramyxoviruses**. A primer extension analysis elucidated the major NP mRNA initiation site with the sequence UCUAAGCC, which showed a moderate homology with the gene-starting consensus sequences of other **paramyxoviruses**. On the other hand, the NP mRNA was terminated at the nucleotide stretch AAAUUCUUUUU, and this sequence was conserved in all the **PIV**-2 genes, indicating that the oligonucleotides will form a part of the gene attenuation signal of **PIV**-2. Comparisons of NP protein sequence indicated a possible subgrouping of the **paramyxoviruses** into two groups, one of which is a group including **PIV**-2, **PIV**-4, MuV, and NDV, and another is a group including **PIV**-3, BPIV-3, and SV. This result supports an idea from our previous studies using polyclonal and monoclonal antibodies. Furthermore, our data indicated that the **PIV**-2 NP protein sequence was more closely related to MV and CDV than to other parainfluenza viruses, **PIV**-3 and SV.

L16 ANSWER 7 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 90357784 MEDLINE
DOCUMENT NUMBER: 90357784 PubMed ID: 2167560
TITLE: Sequence analysis of the phosphoprotein (P) genes of human parainfluenza type 4A and 4B viruses and RNA editing at transcript of the P genes: the number of G residues added is imprecise.
AUTHOR: Kondo K; Bando H; Tsurudome M; Kawano M; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Tokyo, Japan.
SOURCE: VIROLOGY, (1990 Sep) 178 (1) 321-6.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-M55975; GENBANK-M55976
ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 19901026
Last Updated on STN: 20000303
Entered Medline: 19900924

AB We cloned and sequenced the cDNAs against **genomic RNAs** and mRNAs for phosphoproteins (Ps) of human parainfluenza virus types 4A (**PIV**-4A) and 4B (**PIV**-4B). The **PIV**-4A and -4B P genes were 1535 nucleotides including poly(A) tract and were found to have two small open reading frames, neither of which was apparently large enough to encode the P protein. A cluster of G residues was found in **genomic RNA** and the number of G residues was 6 in both **PIV**-4A and -4B. However, the number of G residues at the corresponding site in the mRNAs to the **genomic RNA** was not constant. Three different mRNA cDNA clones were obtained; the first type of mRNA encodes a larger (P) protein of 399 amino acids, the second type encodes V protein of 229 or 230 amino acids, and the third type encodes the smallest protein (156 amino acids). Comparisons on the nucleotide and the amino acid sequences P and V proteins between these two subtypes revealed extensive homologies. However, these homology degrees are lower than that of NP protein. The C-terminal regions of the P and V proteins of **PIV**-4s could be aligned with all other **Paramyxoviruses**, **PIV**-2, mumps virus (MuV), simian virus 5 (SV 5), Newcastle disease virus (NDV), measles virus (MV), canine distemper virus (CDV), Sendai virus (SV), and **PIV**-3. On the other hand, the P-V common (N-terminal) regions showed no homology with MV, CDV, SV, and **PIV**-3. Seven phylogenetic trees of **Paramyxoviruses** were constructed from the entire and partial regions of P and V proteins.

L16 ANSWER 8 OF 10 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 90177230 MEDLINE
DOCUMENT NUMBER: 90177230 PubMed ID: 2155512
TITLE: Molecular cloning and sequence analysis of human parainfluenza type 4A virus HN gene: its irregularities on structure and activities.
AUTHOR: Bando H; Kondo K; Kawano M; Komada H; Tsurudome M; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Japan.
SOURCE: VIROLOGY, (1990 Mar) 175 (1) 307-12.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-M34033
ENTRY MONTH: 199004
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19970203
Entered Medline: 19900404

AB We cloned the cDNA of human parainfluenza type 4A virus (**PIV**-4A) HN gene by reverse-transcription of virus-specific mRNAs and **genomic RNA**, and determined the complete nucleotide sequence of the HN gene. The predicted HN protein sequence of **PIV**-4A showed significant relatedness with those of other **paramyxoviruses**, SV5, NDV, MuV, **PIV**-3, BPIV-3, indicating a common ancestor. The homologies between the viruses suggested that **PIV**-4A is more closely related to NDV, SV5, and MuV than to the parainfluenza viruses, **PIV**-3, bovine parainfluenza type 3 virus (BPIV-3), and Sendai virus (SV). Sixty amino acids were commonly conserved among the viruses, other than **PIV**-4A. Two of these amino acids were substituted in **PIV**-4A HN and are predicted to be located near the active site of the neuraminidase. The analysis of neuraminidase of **PIV**-4 revealed that the activity is hardly detectable, suggesting the significant effect of the substituted amino acid sites on neuraminidase activity.

L16 ANSWER 9 OF 10 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 90357777 MEDLINE
DOCUMENT NUMBER: 90357777 PubMed ID: 2167555
TITLE: Sequence of the fusion protein gene of human parainfluenza type 2 virus and its 3' intergenic region: lack of small hydrophobic (SH) gene.
AUTHOR: Kawano M; Bando H; Ohgimoto S; Kondo K; Tsurudome M; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Tokyo, Japan.
SOURCE: VIROLOGY, (1990 Sep) 178 (1) 289-92.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M55698
ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 19901026
Last Updated on STN: 19970203
Entered Medline: 19900924

AB cDNA clones representing the fusion (F) gene of human parainfluenza virus type 2 (**PIV**-2) were isolated from cDNA libraries constructed from virus-specific mRNA and **genomic RNA**, and the complete nucleotide sequence of the F gene was

determined. The F gene is 1854 nucleotides long and encodes one long open reading frame of 551 amino acids. The cleavage site for activation of the precursor Fo protein is Thr-Arg-Gln-Lys-Arg. The F gene of **PIV**-2 is most closely related to those of simian virus 5 (SV5) and mumps virus (MuV). Interestingly, although the HN glycoprotein of **PIV**-2 shows no relatedness to the HA glycoprotein of measles virus (MV), a distinct homology is found in the F proteins of **PIV**-2 and MV. As concerns F proteins, **paramyxoviruses** can be divided into two subgroups; that is, **PIV**-2, SV5, and MuV belong to one group, and **HPIV** -1, SV, and **PIV**-3 belong to the other group. Newcastle disease virus (NDV) and MV are intermediate. Coding regions for small hydrophobic (SH) proteins have been found between the HN and F genes of SV5 and MuV, which are the viruses most closely related to **PIV**-2. However, such a gene could not be detected in two different strains of **PIV**-2.

L16 ANSWER 10 OF 10 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 90281574 MEDLINE
DOCUMENT NUMBER: 90281574 PubMed ID: 2162103
TITLE: Sequence analysis of P gene of human parainfluenza type 2 virus: P and cysteine-rich proteins are translated by two mRNAs that differ by two nontemplated G residues.
AUTHOR: Ohgimoto S; Bando H; Kawano M; Okamoto K; Kondo K; Tsurudome M; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Japan.
SOURCE: VIROLOGY, (1990 Jul) 177 (1) 116-23.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M37751
ENTRY MONTH: 199007
ENTRY DATE: Entered STN: 19900824
Last Updated on STN: 20000303
Entered Medline: 19900718

AB We cloned and sequenced the cDNAs against **genomic** **RNA** and mRNA for phosphoprotein (P) of human parainfluenza type 2 virus (**PIV**-2). cDNA clone from **genomic** **RNA** was 1439 nucleotides in length excluding poly(A) and was found to have two small open reading frames encoding proteins of 233 and 249 amino acids. Two different mRNA cDNA clones were obtained; that is, one mRNA contained a smaller reading frame coding 225 amino acids, V protein, and the other mRNA contained a larger reading frame coding 395 amino acids, P protein. Both mRNAs had G cluster in

coding frame. The former mRNA contained seven G residues, and two extra G residues were inserted in the latter mRNA. Ten cDNA clones from the **genomic RNA** were identical and were composed of seven G residues, indicating that genomes analyzed here were a homogeneous population. Therefore, V protein is encoded by faithfully copied mRNA and P protein is translated from mRNA in which two additional G residues are nontemplately inserted immediately after seven genomically encoded G residues. The V and P proteins are amino coterminal proteins and have different C termini. The C terminus of V protein is cysteine-rich and bears some resemblance to metal-binding protein of the zinc finger-type motif. P protein sequence of **PIV**-2 showed high homologies with SV 5 (40.4%) and mumps virus (35.5%), and a moderate homology with Newcastle disease virus (20.6%). On the other hand, very little homology was found between **PIV**-2 and other **paramyxoviruses** including Sendai virus, **PIV**-3, and measles virus. The cysteine-rich region in V protein was found to be highly conserved in **PIV**-2, SV 5, and measles virus, suggesting that V protein of **paramyxoviruses** plays important roles in transcription and/or replication. The predicted cysteine-rich V protein was detected in virus-infected cells using antiserum directed against an oligopeptide specific for the predicted V polypeptide.

(FILE 'CAPLUS' ENTERED AT 11:58:41 ON 24 MAY 2001)
L25 11 S (L1 OR L11) AND (NP(S)GENE)
L26 9 S L25 NOT (L10 OR L12)

- Named genes

L26 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:385709 CAPLUS
DOCUMENT NUMBER: 127:1636
TITLE: Preparation of (-)-stranded RNA virus vector having autonomously replicating activity
INVENTOR(S): Nagai, Yoshiyuki; Kato, Atsushi; Murai, Fukashi; Asakawa, Makoto; Sakata, Tsuneaki; Hasegawa, Mamoru; Shioda, Tatsuo
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan; Magai, Yoshiyuki; Kato, Atsushi; Murai, Fukashi; Asakawa, Makoto; Sakata, Tsuneaki; Hasegawa, Mamoru; Shioda, Tatsuo
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9716538	A1	19970509	WO 1996-JP3068	19961022
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI				
CA 2236113	AA	19970509	CA 1996-2236113	19961022
AU 9673351	A1	19970522	AU 1996-73351	19961022
EP 864645	A1	19980916	EP 1996-935402	19961022
R: AT, CH, DE, DK, FR, GB, IT, LI, NL, SE, FI				

PRIORITY APPLN. INFO.: JP 1995-308315 19951031
WO 1996-JP3068 19961022

AB A process for reconstituting virions of Sendai virus by introducing Sendai virus into a host in which early replication genes have been all expressed. The (-)-strand RNA virus vectors exhibits autonomous replication and cell-infection capability, but not propagation capability, are useful for the expression of heterologous gene during, e.g., gene therapy. Also claimed are (1) animal cells expressing Sendai virus **genes** M, **NP**, P/C, L, F, HN, or a subset of them; (2) methods for manufg. heterologous proteins from animal tissue culture fluid or chorioallantoic fluid; and (3) the Sendai virus RNA with defective **gene** M, F, or HN.

L26 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:384291 CAPLUS
DOCUMENT NUMBER: 127:1635
TITLE: Reconstitution of Sendai virus for efficient expression of heterologous genes
INVENTOR(S): Magai, Yoshiyuki; Kato, Atsushi; Murai, Fukashi; Sakata, Tsuneaki; Hasegawa, Mamoru; Shioda, Tatsuo
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan; Magai, Yoshiyuki; Kato, Atsushi; Murai, Fukashi; Sakata, Tsuneaki; Hasegawa, Mamoru; Shioda, Tatsuo
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9716539	A1	19970509	WO 1996-JP3069	19961022

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI

CA 2236378	AA	19970509	CA 1996-2236378	19961022
AU 9673352	A1	19970522	AU 1996-73352	19961022
EP 863202	A1	19980909	EP 1996-935403	19961022
R: AT, CH, DE, DK, FR, GB, IT, LI, NL, SE, FI				
CN 1207123	A	19990203	CN 1996-199467	19961022
CN 1207124	A	19990203	CN 1996-199476	19961022
PRIORITY APPLN. INFO.:			JP 1995-285417	19951101
			WO 1996-JP3069	19961022

AB A method for reconstituting Sendai virions by introducing the genome of Sendai virus into a host cell wherein all of the early replication **genes** (**genes** for **NP**, P/C, and L proteins) are expressed. This method enables gene manipulations of **Sendai virus** and thus makes it possible to utilize **Sendai virus** efficiently as a **vector**. Use of the recombinant Sendai virus for expression of HIV-1 gp120 was demonstrated.

L26 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:30605 CAPLUS
DOCUMENT NUMBER: 124:108004
TITLE: A highly recombinogenic system for the recovery of infectious Sendai paramyxovirus from cDNA: generation of a novel copy-back nondefective interfering virus
AUTHOR(S): Garcin, Dominique; Pelet, Thierry; Calain, Philippe; Roux, Laurent; Curran, Joseph; Kolakofsky, Daniel
CORPORATE SOURCE: Dep. Genetics Microbiol., Univ. Geneva Med Sch., Geneva, CH1211, Switz.
SOURCE: EMBO J. (1995), 14(24), 6087-94
CODEN: EMJODG; ISSN: 0261-4189
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors have recovered infectious Sendai virus (**SeV**) from full-length cDNA (FL-3) by transfecting this cDNA and pGEM plasmids expressing the nucleocapsid protein (NP), phosphoprotein and large proteins into cells infected with a vaccinia virus which expresses T7 RNA polymerase. These cells were then injected into chicken eggs, in which **SeV** grows to very high titers. FL-3 was marked with a BglII site in the leader region and an NsiI site (ATGCAT) in the 5' nontranslated region of the **NP**

gene, creating a new, out-of-frame, 5' proximal AUG. All the virus stocks generated eventually removed this impediment to NP expression, by either point mutation or recombination between FL-3 and pGEM-NP. The recovery system was found to be highly recombinogenic. Even in the absence of selective pressure, one in 20 of the recombinant **SeV** generated had exchanged the **NP gene** of FL-3 with that of pGEM-**NP**. When a fifth plasmid contg. a new genomic 3' end without the presumably deleterious BglII site was included as another target for recombination, the new genomic 3' end was found in the recombinant **SeV** in 12 out of 12 recoveries. Using this approach, a novel copy-back nondefective virus was generated which interferes with wild-type virus replication.

L26 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1995:411035 CAPLUS
DOCUMENT NUMBER: 122:307945
TITLE: Paramyxovirus mRNA editing leads to G deletions as well as insertions
AUTHOR(S): Jacques, Jean-Philippe; Hausmann, Stephane; Kolakofsky, Daniel
CORPORATE SOURCE: School Medicine, University Geneva, Geneva, CH1211, Switz.
SOURCE: EMBO J. (1994), 13(22), 5496-503
CODEN: EMJODG; ISSN: 0261-4189
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Paramyxoviruses are thought to edit their P gene mRNAs co-transcriptionally, by a mechanism in which the polymerase stutters and reads the same template base more than once. Sendai virus (**SeV**) and bovine parainfluenza virus type 3 (bPIV3) are closely related viruses, but **SeV** edits its P gene mRNA with the insertion of a single G residue (at .apprx.59% frequency) within the sequence 5' A6G3, whereas bPIV3 inserts 1 to .apprx.6 Gs at roughly equal frequency within the sequence 5' A6G4. When **SeV** synthetic mini-genomes contg. either **SeV** or bPIV3 P **gene** editing cassettes are expressed from cDNA in cells which are also transfected with the **SeV NP**, P and L **genes**, the virus-specific editing pattern was reproduced in a system that is otherwise completely **SeV**, this suggests that all the information for the virus-specific editing patterns is due to the RNA sequence itself. Unexpectedly, the length of the template C run was found to be crit., even though it varies from 3 go 7 nucleotides in length in different viruses. Expanding this template C run first led to attenuation of the insertions. A stuttering or slippage model to account for these events has been further refined to include a pressure which displaces the nascent strand in a given direction once it has

disengaged from the template, and the similarities of this model to those which account for readthrough of cellular RNA polymerase transcription blocks are discussed.

L26 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1993:442252 CAPLUS
DOCUMENT NUMBER: 119:42252
TITLE: Molecular evolution of human paramyxoviruses. Nucleotide sequence analyses of the human parainfluenza type 1 virus **NP** and M protein **genes** and construction of phylogenetic trees for all the human paramyxoviruses
AUTHOR(S): Miyahara, K.; Kitada, S.; Yoshimoto, M.; Matsumura, H.; Kawano, M.; Komada, H.; Tsurudome, M.; Kusagawa, S.; Nishio, M.; Ito, Y.
CORPORATE SOURCE: Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE: Arch. Virol. (1992), 124(3-4), 255-68
CODEN: ARVIDF; ISSN: 0304-8608
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The nucleotide sequences of the **NP** and M **genes** of human parainfluenza type 1 virus (HPIV-1) were detd. The **NP gene** was 1677 nucleotides long excluding polyadenylic acid. The **NP gene** contained a single large open reading frame (ORF), which encoded a polypeptide of 524 amino acids with a calcd. mol. wt. of 57,736. The M gene 1173 nucleotides long excluding the poly(A) tract and the sequence also contained a single large ORF which encoded a polypeptide of 348 amino acid with a mol. wt. of 38,445, which was inconsistent with 28 kDa previously detd. by SDS-PAGE. The authors aligned the deduced **HPIV**-1 NP and M protein sequences with 12 and 13 other **paramyxoviruses**, resp., suggesting that a common tertiary structure was found in the NPs or Ms of **HPIV**-1 Sendai virus (SV), **HPIV**-3 and BPIV-3 and that other common structure was also maintained in these proteins of **HPIV**-2, SV41 and 5, MuV, **HPIV**-4. Phylogenetic trees were constructed for the NP and M proteins of all the paramyxoviruses of which nucleotide sequences had been previously reported. **Paramyxoviruses** could be subdivided into two groups, i.e., **PIV**-1 group and **PIV**-2 group; the former group is composed of SV, **HPIV**-3 and BPIV-3, and the latter group consists of **HPIV**-2, SV 41, SV 5, MuV, **HPIV**-4A and **HPIV**-4B.

L26 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1992:421419 CAPLUS
DOCUMENT NUMBER: 117:21419

TITLE: RNA packaging signal of human immunodeficiency virus type 1
AUTHOR(S): Hayashi, Takuma; Shioda, Tatsuo; Iwakura, Yoichiro; Shibuta, Hiroshi
CORPORATE SOURCE: Inst. Med. Sci., Univ. Tokyo, Tokyo, 108, Japan
SOURCE: Virology (1992), 188(2), 590-9
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cells infected with a recombinant vaccinia virus carrying the gag and pol regions of the HIV-1 genome (Vac-gag/pol) released HIV-like particles contg. HIV-specific RNA. However, cells infected with another recombinant vaccinia, Vac-gag/pol-dP, derived through the deletion of an 85-base region (nucleotide positions 679-763) of the HIV genome between the primer binding site and the gag initiation codon of Vac-gag/pol, produced HIV-like particles devoid of the HIV-specific RNA. This 85-base deletion was suggested to cause the collapse of a stable stem-loop structure of 46 bases (751-796) around the gag initiation codon. To examine the role of the stem-loop structure in the packaging of RNAs, a vaccinia **vector** plasmid that carried this 46-base sequence followed by the **Sendai virus** nucleocapsid (**NP**) **gene** was constructed. When both Vac-gag/pol-dP and this plasmid were introduced into cells, HIV-like particles released from the cells contained the **NP gene** RNA. However, another vaccinia vector plasmid, which carried the 46-base sequence within the **NP gene**, could not supply RNA for incorporation into HIV-like particles. Computer anal. of this plasmid sequence suggested that the 46-base sequence cannot form the stem-loop structure. These findings suggest that the stem-loop structure formed by the 46-base sequence is crucial as a packaging signal.

L26 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1991:599813 CAPLUS
DOCUMENT NUMBER: 115:199813
TITLE: The nucleoproteins of human parainfluenza virus type 1 and Sendai virus share amino acid sequences and antigenic and structural determinants
AUTHOR(S): Lyn, Deborah; Gill, Dalip S.; Scroggs, Ruth Ann; Portner, Allen
CORPORATE SOURCE: Dep. Virol. Mol. Biol., St. Jude Children's Res. Hosp., Memphis, TN, 38101, USA
SOURCE: J. Gen. Virol. (1991), 72(4), 983-7
CODEN: JGVIAY; ISSN: 0022-1317
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The complete nucleotide sequence of the nucleoprotein (**NP**) **gene** of human parainfluenza virus type 1 (hPIV-1) was detd. from a cDNA clone of mRNA. The mRNA is 1683 nucleotides long (excluding polyadenylic acid) and encodes a protein of 524 amino acids with a predicted Mr of 57548. An amino acid identity of 83% was predicted between the NPs of the human pathogen **hPIV**-1 and the murine **paramyxovirus**, Sendai virus, compared to 72% similarity at the level of the nucleotide sequence. In contrast, the amino acid sequence identity between the NPs of hPIV-1 and hPIV-3 was 59%, suggesting a more distant evolutionary relationship. The NP amino acid sequences of hPIV-1 and Sendai virus were highly conserved in the amino-terminal half of the mol., in which 395 of the first 420 amino acids were identical. Of 11 monoclonal antibodies (MAbs) targeted against the Sendai virus NP, five cross-reacted with the hPIV-1 NP. The MAbs that cross-reacted recognize epitopes within regions of high amino acid similarity between the NPs of the two viruses. Also, five of the eight MAbs raised against hPIV-1 NP cross-reacted with Sendai virus NP. Taken together, these observations suggest that the essential amino acid sequence determinants of the NP structures of hPIV-1 and Sendai virus are conserved despite changes in their nucleotide sequences during evolution. This implies that there was a selective pressure to maintain the important functional domains of the protein.

L26 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1990:230473 CAPLUS
DOCUMENT NUMBER: 112:230473
TITLE: Sequencing analyses and comparison of parainfluenza virus type 4A and 4B **NP** protein **genes**
AUTHOR(S): Kondo, Kunio; Bando, Hisanori; Kawano, Mitsuo; Tsurudome, Masato; Komada, Hiroshi; Nishio, Machiko; Ito, Yasuhiko
CORPORATE SOURCE: Sch. Med., Mie Univ., Tsu, 514, Japan
SOURCE: Virology (1990), 174(1), 1-8
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The nucleotide sequences of the cDNA copies of the mRNA coding for the nucleocapsid proteins (NPs) of human parainfluenza viruses type 4A (PIV-4A) and type 4B (PIV-4B) were detd. The copy of PIV-4A NP mRNA contained 1885 nucleotides encoding a protein with a calcd. mol. wt. of 62,561. The same no. of amino acids with a similar mol. wt. (62,425) were predicted for the PIV-4B NP protein. Comparisons of the nucleotide sequence and the amino acid sequence of NP protein between these 2 subtypes revealed extensive homologies in the nucleotide sequence (87%) and in the amino acid sequence (93%). Furthermore, a conserved region with about 100 amino acids was obsd.

between **PIV**-4s and other **paramyxoviruses**, Newcastle disease virus (NDV), Sendai virus, mumps virus (MuV), **PIV**-3, BPIV-3, measles virus (MV), and canine distemper virus (CDV), indicating a common ancestor for these 9 viruses. The data also indicated that the PIV-4 NP proteins were more closely related to MuV and NDV than to other parainfluenza viruses, PIV-3, BPIV-3, and Sendai virus. Interestingly, the NP protein homol. between PIV-4s and the morbillivirus group, MV and CDV, was slightly higher than that between PIV-4s and the parainfluenza viruses, PIV-3, BPIV-3, and Sendai virus.

L26 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1987:150397 CAPLUS
DOCUMENT NUMBER: 106:150397
TITLE: Complete nucleotide sequence of the matrix protein mRNA and three intergenic junctions of human parainfluenza virus type 3
AUTHOR(S): Luk, Daniel; Masters, Paul S.; Sanchez, Angeles; Banerjee, Amiya K.
CORPORATE SOURCE: Roche Res. Cent., Roche Inst. Mol. Biol., Nutley, NJ, 07110, USA
SOURCE: Virology (1987), 156(1), 189-92
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The complete sequence of the gene encoding the matrix protein (M) of human parainfluenza virus type 3 (PIV-3) was detd. from cDNA clones and from primer extension dideoxy sequencing of the viral genome. The M mRNA is 1150 nucleotides in length, exclusive of polyadenylate, and codes for a protein of 353 amino acids, having a calcd. mol. wt. of 39,480. The M protein of **PIV**-3 was found to have a high degree of sequence homol. with that of a closely related **paramyxovirus**, Sendai virus, and to a lesser extent it contained sequence homol. with two more distant **paramyxoviruses**, measles virus and canine distemper virus. The sequences of the intergenic junctions for the first four **genes** of PIV-3: **NP**, P, M, and F were also detd. Comparison of these sequences yielded a consensus mRNA start sequence of 5'-AGGANNAAAGA-3', an mRNA end sequence of 5'-UAAGAAAAA-3', and an intergenic sequence of 5'-CUU-3'. The end sequence of the M gene is unusual in that it contains an eight base insertion prior to the A5 tract found in the consensus sequence. This disruption appears to cause a high frequency of readthrough by the viral transcriptase at this junction.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 12:09:06 ON 24 MAY 2001)
L27 46 S L25

L28 30 S L27 NOT (L8 OR L15)
L29 11 DUP REM L28 (19 DUPLICATES REMOVED)

L29 ANSWER 1 OF 11 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-402196 [35] WPIDS
DOC. NO. CPI: C2000-121889
TITLE: Recombinant Sendai virus infected egg, used to produce cytokines for use as medicines in the treatment of infectious diseases, tumors, thrombocytopenia, neuropenia and aplastic anemia.
DERWENT CLASS: B04 D16
INVENTOR(S): KAI, C; KATO, A
PATENT ASSIGNEE(S): (NIBI-N) NIPPON BIOCAPITAL LTD; (NIBI-N) NIPPON BIOCAPITAL LTD YG
COUNTRY COUNT: 26
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1013667	A2	20000628	(200035)*	EN	13
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2000201689 A		20000725	(200040)		9

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1013667	A2	EP 1999-402786	19991109
JP 2000201689 A		JP 1999-318550	19991109

PRIORITY APPLN. INFO: JP 1998-317321 19981109
AN 2000-402196 [35] WPIDS
AB EP 1013667 A UPAB: 20000725
NOVELTY - An egg (I) which has been infected with recombinant Sendai virus containing a gene encoding a cytokine, which results in production of the cytokine, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) preparing a cytokine, comprising using (I) and recovering the cytokine; and
(2) a chorioallantoic fluid composition containing a cytokine isolated from (I).
ACTIVITY - Antiinfectious; cytostatic; hemostatic; immunostimulant; antianemic.
MECHANISM OF ACTION - The administered agent is a cytokine.
USE - (I) is used for the production of preferably glycosylated

cytokines, especially gamma -interferon (claimed). (I) can be used to produce other cytokine such as interleukins, colony-stimulating factors, tumor necrosis factors, transforming growth factors, epidermal growth factor, platelet derived growth factor, and hematopoietic factors. These cytokines are useful as medicines, used in the treatment of diseases such as infectious diseases, tumors, thrombocytopenia, neuropenia, and aplastic anemia.

ADVANTAGE - Expressing the **Sendai virus vector** in hen's eggs has been shown to produce proteins having sugar chains similar to those found in mammals, which have been difficult to produce in large amounts in prior art methods. The **Sendai virus** has no pathogeny to humans.

Dwg.0/3

L29 ANSWER 2 OF 11 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:452544 BIOSIS
DOCUMENT NUMBER: PREV200000452544
TITLE: Mapping of domains on the human parainfluenza type 2 virus P and NP proteins that are involved in the interaction with the L protein.
AUTHOR(S): Nishio, Machiko (1); Tsurudome, Masato; Ito, Morihiro; Ito, Yasuhiko
CORPORATE SOURCE: (1) Department of Microbiology, Mie University School of Medicine, 2-174, Edobashi, Tsu-Shi, Mie-Ken, 514-8507 Japan
SOURCE: Virology, (August 1, 2000) Vol. 273, No. 2, pp. 241-247. print.
ISSN: 0042-6822.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Eleven monoclonal antibodies (MAbs) directed against the large (L) protein of human parainfluenza type 2 virus (hPIV-2) were prepared to examine the interactions of the L protein with other viral proteins. Coimmunoprecipitation assays using these MAbs revealed that the L protein directly interacted with the phospho- (P) and nucleocapsid (NP) proteins in vivo and in vitro. Mutational analysis of the P or NP protein was performed to identify the region(s) on these proteins interacting with L protein, indicating that amino acids 278-353 on the P protein and amino acids 403-494 on the NP protein are essential for the binding to the L protein.

L29 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1999:357065 BIOSIS
DOCUMENT NUMBER: PREV199900357065
TITLE: Human parainfluenza virus type 1 matrix and nucleoprotein genes transiently expressed in mammalian cells induce the release of virus-like

particles containing nucleocapsid-like structures.
AUTHOR(S): Coronel, Elizabeth C.; Murti, K. Gopal; Takimoto, Toru; Portner, Allen (1)
CORPORATE SOURCE: (1) Department of Virology and Molecular Biology, St. Jude Children's Research Hospital, 332 N. Lauderdale, Memphis, TN, 38105 USA
SOURCE: Journal of Virology, (Aug., 1999) Vol. 73, No. 8, pp. 7035-7038.
ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The matrix (M) protein plays an essential role in the assembly and budding of some enveloped RNA viruses. We expressed the human parainfluenza virus type 1 (hPIV-1) M and/or **NP genes** into 293T cells using the mammalian expression vector pCAGGS. Biochemical and electron microscopic analyses of transfected cells showed that the M protein alone can induce the budding of virus-like particles (vesicles) from the plasma membrane and that the **NP** protein can assemble into intracellular nucleocapsid-like (NC-like) structures. Furthermore, the coexpression of both the M and **NP genes** resulted in the production of vesicles enclosing NC-like structures, suggesting that the hPIV-1 M protein has the intrinsic ability to induce membrane vesiculation and to incorporate NC-like structures into these budding vesicles.

L29 ANSWER 4 OF 11 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 96134907 MEDLINE
DOCUMENT NUMBER: 96134907 PubMed ID: 8557028
TITLE: A highly recombinogenic system for the recovery of infectious Sendai paramyxovirus from cDNA: generation of a novel copy-back nondefective interfering virus.
AUTHOR: Garcin D; Pelet T; Calain P; Roux L; Curran J; Kolakofsky D
CORPORATE SOURCE: Department of Genetics and Microbiology, University of Geneva Medical School, CMU, Switzerland.
SOURCE: EMBO JOURNAL, (1995 Dec 15) 14 (24) 6087-94.
Journal code: EMB; 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960312
Last Updated on STN: 19960312
Entered Medline: 19960226
AB We have recovered infectious Sendai virus (**SeV**) from

full-length cDNA (FL-3) by transfecting this cDNA and pGEM plasmids expressing the nucleocapsid protein (**NP**), phosphoprotein and large proteins into cells infected with a vaccinia virus which expresses T7 RNA polymerase. These cells were then injected into chicken eggs, in which **SeV** grows to very high titers. FL-3 was marked with a BglII site in the leader region and an NsiI site (ATGCAT) in the 5' nontranslated region of the **NP gene**, creating a new, out-of-frame, 5' proximal AUG. All the virus stocks generated eventually removed this impediment to **NP** expression, by either point mutation or recombination between FL-3 and pGEM-**NP**. The recovery system was found to be highly recombinogenic. Even in the absence of selective pressure, one in 20 of the recombinant **SeV** generated had exchanged the **NP gene** of FL-3 with that of pGEM-**NP**. When a fifth plasmid containing a new genomic 3' end without the presumably deleterious BglII site was included as another target for recombination, the new genomic 3' end was found in the recombinant **SeV** in 12 out of 12 recoveries. Using this approach, a novel copy-back nondefective virus was generated which interferes with wild-type virus replication.

L29 ANSWER 5 OF 11 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 96057576 MEDLINE
DOCUMENT NUMBER: 96057576 PubMed ID: 7559938
TITLE: Expression of Sendai virus nucleocapsid protein in a baculovirus expression system and application to diagnostic assays for Sendai virus infection.
AUTHOR: Wan C H; Riley M I; Hook R R Jr; Franklin C L; Besch-Williford C L; Riley L K
CORPORATE SOURCE: Department of Veterinary Pathology, University of Missouri-Columbia, 65211, USA.
SOURCE: JOURNAL OF CLINICAL MICROBIOLOGY, (1995 Aug) 33 (8) 2007-11.
Journal code: HSH; 7505564. ISSN: 0095-1137.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 19980206
Entered Medline: 19951122

AB The most common diagnostic technique for the detection of **Sendai virus** infection in rodents is serological evaluation by enzyme-linked immunosorbent assay (ELISA) with semipurified preparations of whole virions as antigens. This assay often suffers from a lack of specificity. The goal of the present project was to develop more specific antigens for use in diagnostic

testing by producing recombinant antigens in insect cells. To identify viral proteins immunoreactive in multiple laboratory rodent species, Western blots (immunoblots) of viral polypeptides were probed with immune sera from mice, rats, and hamsters. The nucleocapsid protein (**NP**) reacted with immune sera from all species tested. Therefore, the **NP gene** was selected for cloning and expression in a baculovirus. To construct the recombinant, complementary DNA was synthesized by reverse transcription PCR from **Sendai virus** RNA with primers from the 5' and 3' termini of the **NP**-coding region. Amplified DNA was cloned into a baculovirus transfer **vector** (pBlueBacHis A) and was cotransfected with wild-type baculovirus into insect cells. Baculovirus recombinants containing the **NP gene** were identified by PCR. Evaluation of the recombinant proteins expressed in insect cells by Western blot analysis revealed specific reactivity with immune sera. In comparison with conventional ELISAs that use whole virions as the antigen, ELISAs that use recombinant **NP** were more specific.

L29 ANSWER 6 OF 11 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 95045444 MEDLINE
DOCUMENT NUMBER: 95045444 PubMed ID: 7957115
TITLE: Paramyxovirus mRNA editing leads to G deletions as well as insertions.
AUTHOR: Jacques J P; Hausmann S; Kolakofsky D
CORPORATE SOURCE: Department of Genetics and Microbiology, University of Geneva School of Medicine, Switzerland.
SOURCE: EMBO JOURNAL, (1994 Nov 15) 13 (22) 5496-503.
Journal code: EMB; 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 20000303
Entered Medline: 19941222

AB Paramyxoviruses are thought to edit their P **gene** mRNAs co-transcriptionally, by a mechanism in which the polymerase stutters and reads the same template base more than once. Sendai virus (**SeV**) and bovine parainfluenza virus type 3 (bPIV3) are closely related viruses, but **SeV** edits its P **gene** mRNA with the insertion of a single G residue (at approximately 50% frequency) within the sequence 5' A6G3, whereas bPIV3 inserts 1 to approximately 6 Gs at roughly equal frequency within the sequence 5' A6G4. When **SeV** synthetic mini-genomes containing either **SeV** or bPIV3 P **gene**

editing cassettes are expressed from cDNA in cells which are also transfected with the **SeV NP**, P and L **genes**, the virus-specific editing patterns were reproduced. Since the bPIV3 editing pattern was reproduced in a system that is otherwise completely **SeV**, this suggests that all the information for the virus-specific editing patterns is due to the RNA sequence itself. Unexpectedly, the length of the template C run was found to be critical, even though it varies from 3 to 7 nucleotides in length in different viruses. Expanding this template C run first led to attenuation of the insertion phenotype, and then to deletions rather than insertions. A stuttering or slippage model to account for these events has been further refined to include a pressure which displaces the nascent strand in a given direction once it has disengaged from the template, and the similarities of this model to those which account for readthrough of cellular RNA polymerase transcription blocks are discussed.

L29 ANSWER 7 OF 11 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 92263762 MEDLINE
DOCUMENT NUMBER: 92263762 PubMed ID: 1585635
TITLE: RNA packaging signal of human immunodeficiency virus type 1.
AUTHOR: Hayashi T; Shioda T; Iwakura Y; Shibuta H
CORPORATE SOURCE: Department of Viral Infection, University of Tokyo, Japan.
SOURCE: VIROLOGY, (1992 Jun) 188 (2) 590-9.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199206
ENTRY DATE: Entered STN: 19920626
Last Updated on STN: 19970203
Entered Medline: 19920617

AB Cells infected with a recombinant vaccinia virus carrying the gag and pol regions of the human immunodeficiency virus type 1 genome (Vac-gag/pol) released human immunodeficiency virus (HIV)-like particles containing HIV-specific RNA. However, cells infected with another recombinant vaccinia, Vac-gag/pol-dP, derived through the deletion of an 85-base region (nucleotide positions 679-763) of the HIV genome between the primer binding site and the gag initiation codon of Vac-gag/pol, produced HIV-like particles devoid of the HIV-specific RNA. This 85-base deletion was suggested to cause the collapse of a stable stem-loop structure of 46 bases (751-796) around the gag initiation codon. To examine the role of the stem-loop structure in the packaging of RNAs, we constructed a vaccinia **vector** plasmid that carried this 46-base sequence

followed by the **Sendai virus** nucleocapsid (**NP**) **gene**. When both Vac-gag/pol-dP and this plasmid were introduced into cells, HIV-like particles released from the cells contained the **NP gene** RNA. However, another vaccinia **vector** plasmid, which carried the 46-base sequence in the midst of the **NP gene**, could not supply RNA for incorporation into HIV-like particles. Computer analysis of this plasmid sequence suggested that the 46-base sequence cannot form the stem-loop structure. These findings suggest that the stem-loop structure formed by the 46-base sequence is crucial as a packaging signal.

L29 ANSWER 8 OF 11 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 92296894 MEDLINE
DOCUMENT NUMBER: 92296894 PubMed ID: 1605738
TITLE: Molecular evolution of human paramyxoviruses. Nucleotide sequence analyses of the human parainfluenza type 1 virus **NP** and M protein **genes** and construction of phylogenetic trees for all the human paramyxoviruses.
AUTHOR: Miyahara K; Kitada S; Yoshimoto M; Matsumura H; Kawano M; Komada H; Tsurudome M; Kusagawa S; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Mie, Japan.
SOURCE: ARCHIVES OF VIROLOGY, (1992) 124 (3-4) 255-68. Journal code: 8L7; 7506870. ISSN: 0304-8608.
PUB. COUNTRY: Austria
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L05509; GENBANK-L05510; GENBANK-L05930; GENBANK-S38060; GENBANK-S38067; GENBANK-S45636; GENBANK-S45637; GENBANK-S45639; GENBANK-S45640; GENBANK-S74250
ENTRY MONTH: 199207
ENTRY DATE: Entered STN: 19920724
Last Updated on STN: 19920724
Entered Medline: 19920714

AB The nucleotide sequences of the **NP** and M **genes** of human parainfluenza type 1 virus (**HPIV**-1) were determined. The **NP gene** was 1677 nucleotides long excluding polyadenylic acid. The **NP gene** contained a single large open reading frame (ORF), which encoded a polypeptide of 524 amino acids with a calculated molecular weight of 57,736. The M **gene** 1173 nucleotides long excluding the poly(A) tract and the sequence also contained a single large ORF which encoded a polypeptide of 348 amino acid with a molecular

weight of 38,445, which was inconsistent with 28 kDa previously determined by SDS-PAGE. We aligned the deduced **HPIV**-1 **NP** and M protein sequences with 12 and 13 other **paramyxoviruses**, respectively, suggesting that a common tertiary structure was found in the **NPs** or Ms of **HPIV**-1, Sendai virus (SV), **HPIV**-3 and BPIV-3 and that other common structure was also maintained in these proteins of **HPIV**-2, SV 41 and 5, MuV, **HPIV**-4. Phylogenetic trees were constructed for the **NP** and M proteins of all the **paramyxoviruses** of which nucleotide sequences had been previously reported. **Paramyxoviruses** could be subdivided into two groups, i.e., **PIV**-1 group and **PIV**-2 group; the former group is composed of **HPIV**-1, SV, **HPIV**-3 and BPIV-3, and the latter group consists of **HPIV**-2, SV 41, SV 5, MuV, **HPIV**-4 A and **HPIV**-4 B.

L29 ANSWER 9 OF 11 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 91202138 MEDLINE
DOCUMENT NUMBER: 91202138 PubMed ID: 1707951
TITLE: The nucleoproteins of human parainfluenza virus type 1 and Sendai virus share amino acid sequences and antigenic and structural determinants.
AUTHOR: Lyn D; Gill D S; Scroggs R A; Portner A
CORPORATE SOURCE: Department of Virology and Molecular Biology, St Jude Children's Research Hospital, Memphis, Tennessee 38101.
CONTRACT NUMBER: AI-05343 (NIAID)
AI-11949 (NIAID)
CA-21765 (NCI)
SOURCE: JOURNAL OF GENERAL VIROLOGY, (1991 Apr) 72 (Pt 4) 983-7.
Journal code: I9B; 0077340. ISSN: 0022-1317.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D01019; GENBANK-D01056; GENBANK-D01070; GENBANK-D10141; GENBANK-D10143; GENBANK-D10144; GENBANK-D10145; GENBANK-D10146; GENBANK-D10148; GENBANK-D10149
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 19910607
Last Updated on STN: 19970203
Entered Medline: 19910523

AB The complete nucleotide sequence of the nucleoprotein (**NP**) **gene** of human parainfluenza virus type 1 (**hPIV**-1) was determined from a cDNA clone of mRNA. The mRNA is 1683

nucleotides long (excluding polyadenylic acid) and encodes a protein of 524 amino acids with a predicted Mr of 57,548. An amino acid identity of 83% was predicted between the **NPs** of the human pathogen **hPIV**-1 and the murine **paramyxovirus**, Sendai virus, compared to 72% similarity at the level of the nucleotide sequence. In contrast, the amino acid sequence identity between the **NPs** of **hPIV**-1 and **hPIV**-3 was 59%, suggesting a more distant evolutionary relationship. The **NP** amino acid sequences of **hPIV**-1 and Sendai virus were highly conserved in the amino-terminal half of the molecule, in which 395 of the first 420 amino acids were identical. Of 11 monoclonal antibodies (MAbs) targeted against the Sendai virus **NP**, five cross-reacted with the **hPIV**-1 **NP** . The MAbs that cross-reacted recognize epitopes within regions of high amino acid similarity between the **NPs** of the two viruses. Also, five of the eight MAbs raised against **hPIV** -1 **NP** cross-reacted with Sendai virus **NP**. Taken together, our observations suggest that the essential amino acid sequence determinants of the **NP** structures of **hPIV** -1 and Sendai virus are conserved despite changes in their nucleotide sequences during evolution. This implies that there was a selective pressure to maintain the important functional domains of the protein.

L29 ANSWER 10 OF 11 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 90101365 MEDLINE
DOCUMENT NUMBER: 90101365 PubMed ID: 2152988
TITLE: Sequencing analyses and comparison of parainfluenza virus type 4A and 4B **NP** protein **genes**.
AUTHOR: Kondo K; Bando H; Kawano M; Tsurudome M; Komada H; Nishio M; Ito Y
CORPORATE SOURCE: Department of Microbiology, Mie University School of Medicine, Japan.
SOURCE: VIROLOGY, (1990 Jan) 174 (1) 1-8.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M32982; GENBANK-M32983
ENTRY MONTH: 199002
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19980206
Entered Medline: 19900205

AB The nucleotide sequences of the cDNA copies of the mRNA coding for the nucleocapsid proteins (NPs) of human parainfluenza viruses type 4A (**PIV**-4A) and type 4B (**PIV**-4B) were

determined. The copy of **PIV**-4A NP mRNA contained 1885 nucleotides encoding a protein with a calculated molecular weight of 62,561. The same number of amino acids with a similar molecular weight (62,425) were predicted for the **PIV**-4B NP protein. Comparisons on the nucleotide sequence and the amino acid sequence of NP protein between these two subtypes revealed extensive homologies in the nucleotide sequence (87%) and in the amino acid sequence (93%). Furthermore, a conserved region with about 100 amino acids was observed between **PIV**-4s and other **paramyxoviruses**, Newcastle disease virus (NDV), Sendai virus, mumps virus (MuV), **PIV**-3, BPIV-3, measles virus (MV), and canine distemper virus (CDV), indicating a common ancestor for these nine viruses. Our data also indicated that the **PIV** -4 NP proteins were more closely related to MuV and NDV than to other parainfluenza viruses, **PIV**-3, BPIV-3, and Sendai virus. Interestingly, the NP protein homology between **PIV** -4s and the morbillivirus group, MV and CDV, was slightly higher than that between **PIV**-4s and the parainfluenza viruses, **PIV**-3, BPIV-3, and Sendai virus.

L29 ANSWER 11 OF 11 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 87122153 MEDLINE
DOCUMENT NUMBER: 87122153 PubMed ID: 3027966
TITLE: Complete nucleotide sequence of the matrix protein mRNA and three intergenic junctions of human parainfluenza virus type 3.
AUTHOR: Luk D; Masters P S; Sanchez A; Banerjee A K
SOURCE: VIROLOGY, (1987 Jan) 156 (1) 189-92.
Journal code: XEA; 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M16569
ENTRY MONTH: 198703
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870302

AB The complete sequence of the **gene** encoding the matrix protein (M) of human parainfluenza virus type 3 (**PIV**-3) was determined from cDNA clones and from primer extension dideoxy sequencing of the viral genome. The M mRNA is 1150 nucleotides in length, exclusive of polyadenylate, and codes for a protein of 353 amino acids, having a calculated molecular weight of 39,480. The M protein of **PIV**-3 was found to have a high degree of sequence homology with that of a closely related **paramyxovirus**, Sendai virus, and to a lesser extent it contained sequence homology with two more distant

paramyxoviruses, measles virus and canine distemper virus. We also determined the sequences of the intergenic junctions for the first four **genes** of **PIV-3**: **NP**, P, M, and F. Comparison of these sequences yielded a consensus mRNA start sequence of 5'-AGGANNAAAGA-3', an mRNA end sequence of 5'-UAAGAAAAA-3', and an intergenic sequence of 5'-CUU-3'. The end sequence of the M **gene** is unusual in that it contains an eight base insertion prior to the A5 tract found in the consensus sequence. This disruption appears to cause a high frequency of readthrough by the viral transcriptase at this junction.

(FILE 'MEDLINE' ENTERED AT 12:10:49 ON 24 MAY 2001)

L30 1956 SEA FILE=MEDLINE ABB=ON PLU=ON PARAMYXOVIRUS/CT
L32 46103 SEA FILE=MEDLINE ABB=ON PLU=ON RNA/CT
L34 11 SEA FILE=MEDLINE ABB=ON PLU=ON L30 AND L32

L34 ANSWER 1 OF 11 MEDLINE
AN 1998010794 MEDLINE
TI [A simple method for RNA isolation and purification].
Prostol Metod Vydeleniya i Ochistki RNK.
AU Gribanov O G; Shcherbakov A V; Perevozchikova N A; Drygin V V; Gusev A A
SO BIOORGANICHESKAIA KHIMIIA, (1997 Sep) 23 (9) 763-5.
Journal code: 9Z8; 7804941. ISSN: 0132-3423.
AB RNAs from Escherichia coli cells, Syrian hamster kidney cells, foot-and-mouth disease virus, and Newcastle disease virus were isolated using glass fiber filters GF/F or GF/C. The RNA was reversibly adsorbed on the filters in the presence of 2 M guanidine thiocyanate and 50% ethanol (or isopropanol) and eluted with water. The fraction composition of the isolated RNA depended on the guanidine thiocyanate and alcohol concentrations in the adsorption and washing procedures. The RNA preparations obtained by this method can be used in reverse transcription and reverse transcription-polymerase chain reaction without additional purification.

L34 ANSWER 2 OF 11 MEDLINE
AN 86124713 MEDLINE
TI Molecular cloning and sequence analysis of the human parainfluenza 3 virus RNA encoding the nucleocapsid protein.
AU Galinski M S; Mink M A; Lambert D M; Wechsler S L; Pons M W
SO VIROLOGY, (1986 Mar) 149 (2) 139-51.
Journal code: XEA; 0110674. ISSN: 0042-6822.
AB The sequence of 1690 nucleotides from the 5' end of the viral complementary RNA for the human parainfluenza 3 virus was determined by molecular cloning. One large open reading frame consisting of 1548 nucleotides was demonstrated. The encoded protein, the nucleocapsid protein (NP), consists of 515 amino acids, and has a

predicted molecular weight of 57,819. A noncoding 5' sequence of 51 nucleotides is present at the end of the NP-mRNA. Two consensus sequences were identified which are homologous with sequences found in Sendai virus. One of these sequences, AGGATTAAAG, was located at the 5' end of the nucleocapsid mRNA and may function in transcription initiation. The other consensus sequence, GTAAGGGAA, was found in the viral genomic leader sequence. The nucleocapsid protein amino acid sequence was compared to other members of the Paramyxoviridae family. The parainfluenza 3 virus protein nucleocapsid amino acid sequence demonstrated a high degree of homology with the Sendai virus nucleocapsid protein. Seventy percent of the first 387 amino acids from the amino termini were identical. Little homology was observed in the distal carboxy termini.

L34 ANSWER 3 OF 11 MEDLINE
AN 80054161 MEDLINE
TI Characterization of bovine parainfluenza virus type 3.
AU Shibuta H; Kanda T; Adachi A; Yogo Y
SO MICROBIOLOGY AND IMMUNOLOGY, (1979) 23 (7) 617-28.
Journal code: MX7; 7703966. ISSN: 0385-5600.
AB Bovine parainfluenza virus type 3 (PIV-3) has a buoyant density of 1.197. The RNA of PIV-3, like that of Sendai virus, is a single continuous chain which lacks polyadenylic acid sequences and tends to self-anneal to a marked extent. It has a sedimentation coefficient of 42S and a molecular weight of 4.5 X 10(6), being slightly smaller than Sendai virus RNA (47S, 5.3 X 10(6)). PIV-3 has 5 main structural proteins, of which 2 are glycoproteins. The molecular weights of protein 1, protein 2, protein 3, glycoprotein 1, and glycoprotein 2 were estimated to be 79,000, 68,000, 35,000, 69,000, and 55,000, respectively. Protein 2 was suggested to be nucleocapsid protein.

L34 ANSWER 4 OF 11 MEDLINE
AN 77030062 MEDLINE
TI The assessment in sheep of an inactivated vaccine of parainfluenza 3 virus incorporating double stranded RNA (BRL 5907) as adjuvant.
AU Wells P W; Sharp J M; Burrells C; Rushton B; Smith W D
SO JOURNAL OF HYGIENE, (1976 Oct) 77 (2) 255-61.
Journal code: IEF; 0375374. ISSN: 0022-1724.
AB The serological responses of conventionally reared sheep were compared after vaccination with inactivated parainfluenza 3 (PI3) virus incorporated in three different adjuvants. Inactivated PI3 virus with the double-stranded RNA, BRL 5907 in an oil emulsion was shown to stimulate higher serum antibody titres over the first 5 weeks after vaccination than virus with and without BCG emulsified in oil. The ability of this vaccine to protect specific pathogen-free lambs against challenge with PI3 virus was examined in a second experiment. In this experiment the vaccine stimulated virus

neutralizing and haemagglutination inhibiting antibodies in the serum. After intranasal and intratracheal inoculation with PI3 virus at challenge, vaccinated lambs showed no clinical illness and virus isolation was confined, except in one lamb, to the first two days. In contrast, unvaccinated lambs developed respiratory disease and virus was isolated daily for 7 days after challenge.

L34 ANSWER 5 OF 11 MEDLINE
AN 72239525 MEDLINE
TI Influence of nitrogen dioxide on the uptake of parainfluenza-3 virus by alveolar macrophages.
AU Williams R D; Acton J D; Myrvik Q N
SO JOURNAL OF THE RETICULOENDOTHELIAL SOCIETY, (1972 Jun) 11 (6) 627-36.
Journal code: JWV; 0206462. ISSN: 0033-6890.

L34 ANSWER 6 OF 11 MEDLINE
AN 70200896 MEDLINE
TI [Ribonucleic acid of parainfluenza vius type 3].
L'acide ribonucleique du paramyxovirus parainfluenzae, type 3.
AU Dubois M F; Daniel P; Mehran A R
SO ARCHIV FUR DIE GESAMTE VIRUSFORSCHUNG, (1970) 29 (4) 307-14.
Journal code: 73K; 7506868. ISSN: 0003-9012.

L34 ANSWER 7 OF 11 MEDLINE
AN 70042400 MEDLINE
TI Inducers of interferon and host resistance. VII. Antiviral efficacy of double-stranded RNA of natural origin.
AU Nemes M M; Tytell A A; Lampson G P; Field A K; Hilleman M R
SO PROCEEDINGS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE, (1969 Nov) 132 (2) 784-9.
Journal code: PXZ; 7505892. ISSN: 0037-9727.

L34 ANSWER 8 OF 11 MEDLINE
AN 69241312 MEDLINE
TI Adaptation of the parainfluenza 3(EA--106) virus to a new host. I. Adaptation of the strain from tissue lines to the chick embryo.
AU Lobodzinska M; Sypulowa A; Skurska Z
SO ARCHIVUM IMMUNOLOGIAE ET THERAPIAE EXPERIMENTALIS, (1969) 17 (2) 164-73.
Journal code: 79O; 0114365. ISSN: 0004-069X.

L34 ANSWER 9 OF 11 MEDLINE
AN 69232191 MEDLINE
TI Sensitivity of common respiratory viruses to an interferon inducer in human cells.
AU Hill D A; Baron S; Chanock R M
SO LANCET, (1969 Jul 26) 2 (7613) 187-8.

Journal code: L0S; 2985213R. ISSN: 0140-6736.

L34 ANSWER 10 OF 11 MEDLINE
AN 68052107 MEDLINE
TI The effects of substituted benzimidazoles on the growth of viruses and the nucleic acid metabolism of host cells.
AU Bucknall R A
SO JOURNAL OF GENERAL VIROLOGY, (1967 Jan) 1 (1) 89-99.
Journal code: I9B; 0077340. ISSN: 0022-1317.

L34 ANSWER 11 OF 11 MEDLINE
AN 66121041 MEDLINE
TI Artificial heterokaryons of animal cells from different species.
AU Harris H; Watkins J F; Ford C E; Schoefl G I
SO JOURNAL OF CELL SCIENCE, (1966 Mar) 1 (1) 1-30.
Journal code: HNK; 0052457. ISSN: 0021-9533.

(FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 12:13:08 ON 24 MAY 2001)

L35	211	S	(TOKOSUMI T? OR TSUYOSHI T?)/AU
L36	3094	S	(IIDA A? OR AKIHIRO I?)/AU
L37	20093	S	(MAMORU H? OR HASEGAWA M?)/AU
L38	12360	S	(NAGAI Y? OR YOSHIYUKI N?)/AU
L39	0	S	L35 AND L36 AND L37 AND L38
L40	0	S	L35 AND (L36 OR L37 OR L38)
L41	9	S	L36 AND (L37 OR L38)
L42	53	S	L37 AND L38
L43	35696	S	L35 OR L36 OR L37 OR L38
L44	115	S	(L42 OR L43) AND (L1 OR L11)
L45	72	S	L44 AND (RNA OR RIBONUCLEIC)
L46	74	S	L41 OR L45
L47	26	DUP REM	L46 (48 DUPLICATES REMOVED)

_ Author(s)

L47 ANSWER 1 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 1
ACCESSION NUMBER: 2001:240514 CAPLUS
TITLE: Y2, the smallest of the Sendai virus C proteins, is fully capable of both counteracting the antiviral action of interferons and inhibiting viral **RNA** synthesis
AUTHOR(S): Kato, Atsushi; Ohnishi, Yukano; Kohase, Masayoshi; Saito, Sakura; Tashiro, Masato; **Nagai, Yoshiyuki**
CORPORATE SOURCE: Department of Viral Diseases and Vaccine Control, National Institute of Infectious Diseases, Tokyo, 208-0011, Japan
SOURCE: J. Virol. (2001), 75(8), 3802-3810
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal
LANGUAGE: English

AB An open reading frame (ORF) overlapping the amino-terminal portion of the Sendai virus (**SeV**) P ORF in the +1 frame produces a nested set of carboxy-coterminal proteins, C', C, Y1, and Y2, which are referred to collectively as the C proteins. The C proteins are extremely versatile triple-role players; they counteract the antiviral action of interferons (IFNs), inhibit viral **RNA** synthesis, and are involved in virus assembly. In this study, we established HeLa cell lines stably expressing the C, Y1, and Y2 proteins individually and examd. the capacities of these cells to circumvent the antiviral action of alpha/beta IFN (IFN-.alpha./.beta.) and IFN-.gamma. and to inhibit viral transcription. The assay protocols included monitoring of IFN-.alpha./.beta.-mediated signaling by interferon-stimulated response element-driven reporter gene expression and of the antiviral state induced by IFN-.alpha./.beta. and IFN-.gamma. and measurement of reporter gene expression from an **SeV** minigenome, as well as quantification of **SeV** primary transcripts. When necessary, the activities measured were carefully normalized to the expression levels of the resp. C proteins in cells. The data obtained clearly indicate that the smallest protein, Y2, was as active as the C and Y1 proteins in both counteracting the antiviral action of IFNs and inhibiting viral transcription. The data further show that intracellular transexpression of either C, Y1, or Y2 rendered HeLa cells moderately or only poorly permissive for not only wild-type **SeV** but also 4C(-) **SeV**, which expressed none of the four C proteins. On the basis of these findings, the roles of **SeV** C proteins in the natural life cycle are discussed.

REFERENCE COUNT: 47
REFERENCE(S):
(1) Atreya, P; Virology 1999, V261, P227 CAPLUS
(2) Cadd, T; J Virol 1996, V70, P5067 CAPLUS
(3) Chin, Y; Mol Cell Biol 1997, V17, P5328 CAPLUS
(4) Curran, J; Virology 1992, V189, P647 CAPLUS
(5) Didcock, L; J Virol 1999, V73, P3125 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 2 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 2
ACCESSION NUMBER: 2000:824432 CAPLUS
DOCUMENT NUMBER: 134:15152
TITLE: Paramyxoviridae virus vector defective in envelope gene F and its construction
INVENTOR(S): Li, Hai-Ou; Shu, Tsugumine; Kuma, Hidekazu; Ueda, Yasuji; Asakawa, Makoto; **Hasegawa, Mamoru**; **Iida, Akihiro**; Tokitou, Fumino; Hirata, Takahiro; Tokusumi, Tsuyoshi

PATENT ASSIGNEE(S): DNAVEC Research Inc., Japan
SOURCE: PCT Int. Appl., 177 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000070070	A1	20001123	WO 2000-JP3195	20000518

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 1999-200739 A 19990518

AB Virus virions defective in F gene are successfully collected by using a Sendai virus genomic cDNA with deletion of F gene. Further, infectious viral particles defective in F gene are successfully constructed by using F-expression cells as helper cells. Also, virus virions defective in F gene and HN gene are successfully collected by using a virus genomic cDNA with deletion of both of F gene and HN gene. Further, infectious viral particles defective in F gene and HN gene are successfully produced by using F- and HN-expression cells as helper cells. A virus being defective in F gene and HN gene and having F protein is constructed by using F-expression cells as helper cells. Further, a VSV-G pseudo type virus is successfully constructed by using VSV-G-expression cells. Techniques for constructing these defective viruses contribute to the development of vectors of Paramyxoviridae usable in gene therapy.

REFERENCE COUNT: 4
REFERENCE(S):
(1) Bitzer, M; J Virol 1997, V71(7), P5481 CAPLUS
(2) Hai-Ou LI; J Virol 2000, V74(14), P6564
(3) Kido, H; Biopolymers (Peptide Science) 1999, V51(1), P79 CAPLUS
(4) Yu, D; Genes to Cells 1997, V2(7), P457 CAPLUS

L47 ANSWER 3 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 3
ACCESSION NUMBER: 2000:824418 CAPLUS
DOCUMENT NUMBER: 134:2564

TITLE: RNP originating in **paramyxovirus** and use for preparation of safe viral **vectors**
INVENTOR(S): Li, Hai-Ou; Shu, Tsugumine; Kuma, Hidekazu; Ueda, Yasuji; Asakawa, Makoto; **Hasegawa, Mamoru**; **Iida, Akihiro**; Hirata, Takahiro
PATENT ASSIGNEE(S): DNAVEC Research Inc., Japan
SOURCE: PCT Int. Appl., 172 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000070055	A1	20001123	WO 2000-JP3194	20000518

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 1999-200740 A 19990518

AB A functional RNP (ribonucleoprotein complex) contg. (-)-chain single stranded **RNA** originating in Sendai virus which has been modified so as not to express any envelope protein. An RNP contg. this foreign gene is prepd. and inserted into a cell with the use of a cationic liposome, thereby successfully expressing the foreign gene. Vectors derived from the NS-**RNA** (minus-stranded **RNA**) virus are deemed safe for use.

REFERENCE COUNT: 2
REFERENCE(S):
(1) Huntley, C; J Biol Chem 1997, V272(26), P16578 CAPLUS
(2) Rhirnov, O; Virology 1990, V176(1), P274

L47 ANSWER 4 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 4
ACCESSION NUMBER: 2000:133839 CAPLUS
DOCUMENT NUMBER: 132:190468
TITLE: **RNA** virus **vector** having contact infiltration capability with **Sendai virus** M protein deficiency or defect
INVENTOR(S): Asakawa, Makoto; **Hasegawa, Mamoru**

PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000009700	A1	20000224	WO 1999-JP4333	19990810
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9950675	A1	20000306	AU 1999-50675	19990810

PRIORITY APPLN. INFO.: JP 1998-227398 A 19980811
WO 1999-JP4333 W 19990810

AB An **RNA** virus vector with capabilities for infection, autonomous replication, and contact infiltration but no communicability, and its method of synthesis are provided. The matrix protein (M protein) gene of non-segmented (-) strand virus, Sendai virus, origin is either missing or defective in the **RNA**. The template DNA capable of transcribing such **RNA** in vitro or in vivo, and a method of expressing exogenous gene in mammals using the **RNA** virus are also claimed. M protein deficient and defective vectors had a capability to form plaques smaller than the wild type, but did not multiply within chicken egg. The plaques due to M protein deficient and defective **vectors** were immunostained with anti-**Sendai virus** antibody, but not with anti-M protein monoclonal antibody, suggesting that they lacked normal M protein.

REFERENCE COUNT: 4
REFERENCE(S):
(1) Dnavec Research Inc; EP 864645 A CAPLUS
(2) Dnavec Research Inc; WO 9716538 A1 1997 CAPLUS
(3) Kondou, T; The Journal of Biological Chemistry 1993, V268(29), P21924
(4) Mottet, G; Virology 1996, V221(1), P159 CAPLUS

L47 ANSWER 5 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 5
ACCESSION NUMBER: 2000:34991 CAPLUS

DOCUMENT NUMBER: 132:74561
TITLE: Nerve cells-specific gene transfer using (-)-strand **RNA** virus vector
INVENTOR(S): Fukumura, Masayuki; Asakawa, Makoto; **Hasegawa, Mamoru**
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000001837	A1	20000113	WO 1999-JP3552	19990701
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9943955	A1	20000124	AU 1999-43955	19990701
EP 1094115	A1	20010425	EP 1999-926878	19990701
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: JP 1998-204333 A 19980703
WO 1999-JP3552 W 19990701

AB A recombinant **vector** derived from a (-)-strand **RNA** virus, such as **Sendai virus** (**SeV**) of **Paramyxoviridae**, is used for nerve cells-specific gene transfer for gene therapy. The method was demonstrated by introducing the gene for green fluorescent protein (GFP) into the cultured nerve cell lines, primary nerve cell culture, or cerebral ventricles of rat or mice. Expression of the gene for FGF-1 or FGF-5 from vector FGF-1-**SeV** or FGF-5-**SeV** inoculated into the cerebral ventricles of mice, and their effects on feed redn. were shown. Hippocampus ependymal cell.

REFERENCE COUNT: 2
REFERENCE(S):
(1) Christopher, M; Neurochemical Research 1993, V18(10), P1089
(2) Nakanishi, M; Journal of Controlled Release 1998, V54(1), P61 CAPLUS

L47 ANSWER 6 OF 26 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 2000:656331 SCISEARCH
THE GENUINE ARTICLE: 346JD
TITLE: Involvement of the zinc-binding capacity of Sendai virus V protein in viral pathogenesis
AUTHOR: Huang C; Kiyotani K; Fujii Y; Fukuhara N; Kato A; **Nagai Y**; Yoshida T; Sakaguchi T (Reprint)
CORPORATE SOURCE: HIROSHIMA UNIV, SCH MED, DEPT BACTERIOL, MINAMI KU, 1-2-3 KASUMI, HIROSHIMA 7348551, JAPAN (Reprint); HIROSHIMA UNIV, SCH MED, DEPT BACTERIOL, MINAMI KU, HIROSHIMA 7348551, JAPAN; UNIV TOKYO, INST MED SCI, DEPT VIRAL INFECT, TOKYO 1088639, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: JOURNAL OF VIROLOGY, (SEP 2000) Vol. 74, No. 17, pp. 7834-7841.
Publisher: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904.
ISSN: 0022-538X.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 31

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The V protein of Sendai virus (**SeV**) is nonessential to virus replication in cell culture but indispensable to viral pathogenicity in mice. The highly conserved cysteine-rich zinc finger-like domain in its carboxyl terminus is believed to be responsible for this viral pathogenicity. in the present study, we showed that the cysteine-rich domain of the **SeV** V protein could actually bind zinc by using glutathione-S-transferase fusion proteins. When the seven conserved cysteine residues at positions 337, 341, 353, 355, 358, 362, and 365 were replaced individually, the zinc-binding capacities of the mutant proteins were greatly impaired, ranging from 22 to 68% of that of the wild type. We then recovered two mutant **SeVs** from cDNA, which have V-C341S and V-C365R mutations and represent maximal and minimal zinc-binding capacities among the corresponding mutant Fusion proteins, respectively. The mutant viruses showed viral protein synthesis and growth patterns similar to those of wild-type **SeV** in cultured cells. However, the mutant viruses were strongly attenuated in mice in a way similar to that of **SeV** V-Delta C, which has a truncated V protein lacking the cysteine-rich domain, by exhibiting earlier viral clearance from the mouse lung and less virulence to mice. We therefore conclude that the zinc-binding capacity of the V protein is involved in viral pathogenesis.

L47 ANSWER 7 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 6
ACCESSION NUMBER: 2000:466593 CAPLUS
DOCUMENT NUMBER: 133:345261

TITLE: A cytoplasmic **RNA vector** derived from nontransmissible **sendai virus** with efficient gene transfer and expression

AUTHOR(S): Li, Hai-Ou; Zhu, Ya-Feng; Asakawa, Makoto; Kuma, Hidekazu; Hirata, Takahiro; Ueda, Yasuji; Lee, Yun-Sik; Fukumura, Masayuki; **Iida, Akihiro**; Kato, Atsushi; **Nagai, Yoshiyuki**; **Hasegawa, Mamoru**

CORPORATE SOURCE: DNAVEC Research Inc., Tsukuba, 305-0856, Japan

SOURCE: J. Virol. (2000), 74(14), 6564-6569
CODEN: JOVIAM; ISSN: 0022-538X

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We have recovered a virion from defective cDNA of Sendai virus (**SeV**) that is capable of self-replication but incapable of transmissible-virion prodn. This virion delivers and expresses foreign genes in infected cells, and this is the first report of a gene expression **vector** derived from a defective viral genome of the **Paramyxoviridae**. First, functional ribonucleoprotein complexes (RNPs) were recovered from **SeV** cloned cDNA defective in the F (envelope fusion protein) gene, in the presence of plasmids expressing nucleocapsid protein and viral **RNA** polymerase. Then the RNPs were transfected to the cells inducibly expressing F protein. Virion-like particles thus obtained had a titer of 0.5 .times. 108 to 1.0 .times. 108 cell infectious units/mL and contained F-defective **RNA** genome. This defective vector amplified specifically in an F-expressing packaging cell line in a trypsin-dependent manner but did not spread to F-nonexpressing cells. This vector infected and expressed an enhanced green fluorescent protein reporter gene in various types of animal and human cells, including nondividing cells, with high efficiency. These results suggest that this vector has great potential for use in human gene therapy and vaccine delivery systems.

REFERENCE COUNT: 36

REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 8 OF 26 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 2000283821 MEDLINE

DOCUMENT NUMBER: 20283821 PubMed ID: 10823869
TITLE: Versatility of the accessory C proteins of Sendai virus: contribution to virus assembly as an additional role.
AUTHOR: Hasan M K; Kato A; Muranaka M; Yamaguchi R; Sakai Y; Hatano I; Tashiro M; **Nagai Y**
CORPORATE SOURCE: Department of Viral Diseases and Vaccine Control, National Institute of Infectious Diseases, Tokyo 208-0011, Japan.
SOURCE: JOURNAL OF VIROLOGY, (2000 Jun) 74 (12) 5619-28.
Journal code: KCV; 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000706
Last Updated on STN: 20000706
Entered Medline: 20000629

AB The P/C mRNA of Sendai virus (**SeV**) encodes a nested set of accessory proteins, C', C, Y1, and Y2, referred to collectively as C proteins, using the +1 frame relative to the open reading frame of phospho (P) protein and initiation codons at different positions. The C proteins appear to be basically nonstructural proteins as they are found abundantly in infected cells but greatly underrepresented in the virions. We previously created a 4C(-) **SeV**, which expresses none of the four C proteins, and concluded that the C proteins are categorically nonessential gene products but greatly contribute to viral full replication and infectivity (A. Kurotani et al., Genes Cells 3:111-124, 1998). Here, we further characterized the 4C(-) virus multiplication in cultured cells. The viral protein and mRNA synthesis was enhanced with the mutant virus relative to the parental wild-type (WT) **SeV**. However, the viral yields were greatly reduced. In addition, the 4C(-) virions appeared to be highly anomalous in size, shape, and sedimentation profile in a sucrose gradient and exhibited the ratios of infectivity to hemagglutination units significantly lower than those of the WT. In the WT infected cells, C proteins appeared to colocalize almost perfectly with the matrix (M) proteins, pretty well with an external envelope glycoprotein (hemagglutinin-neuraminidase [HN]), and very poorly with the internal P protein. In the absence of C proteins, there was a significant delay of the incorporation of M protein and both of the envelope proteins, HN and fusion (F) proteins, into progeny virions. These results strongly suggest that the accessory and basically nonstructural C proteins are critically required in the **SeV** assembly process. This role of C proteins was further found to be independent of their recently discovered function to counteract the antiviral action of interferon-

alpha/beta. **SeV** C proteins thus appear to be quite versatile.

L47 ANSWER 9 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 8
ACCESSION NUMBER: 2000:469082 CAPLUS
DOCUMENT NUMBER: 133:191330
TITLE: Viral mutation accelerated by nitric oxide production during infection in vivo
AUTHOR(S): Akaike, Takaaki; Fujii, Shigemoto; Kato, Atsushi; Yoshitake, Jun; Miyamoto, Yoichi; Sawa, Tomohiro; Okamoto, Shinichiro; Suga, Moritaka; Asakawa, Makoto; **Nagai, Yoshiyuki**; Maeda, Hiroshi
CORPORATE SOURCE: Departments of Microbiology and Medicine I, Kumamoto University School of Medicine, Kumamoto, 860-0811, Japan
SOURCE: FASEB J. (2000), 14(10), 1447-1454
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nitric oxide (NO), superoxide (O2-), and their reaction product peroxynitrite (ONOO-) are generated in excess during a host's response against viral infection, and contribute to viral pathogenesis by promoting oxidative stress and tissue injury. Here we demonstrate that NO and peroxynitrite greatly accelerates the mutation of Sendai virus (**SeV**), a nonsegmented neg.-strand **RNA** virus, by using green fluorescent protein (GFP) inserted into and expressed by a recombinant **SeV** (GFP-**SeV**) as an indicator for mutation. GFP-**SeV** mutation frequencies were much higher in the wild-type mice than in those lacking inducible NO synthase, suggesting that mutation of the virus in vivo is NO dependent. High levels of NO and NO-mediated oxidative stress were induced by GFP-**SeV** infection in the lung of the wild-type mice, but not in the iNOS-deficient mice, as evidenced by ESR spectroscopy and immunohistochem. anal. for nitrotyrosine formation as well as histopathol. examn. Furthermore, peroxynitrite, an NO-derived reactive nitrogen intermediate, enhanced viral mutation in vitro. These results indicate that the oxidative stress induced by NO produced during the natural course of viral infection increases mutation, expands the quasispecies spectrum, and facilitates evolution of **RNA** viruses.

REFERENCE COUNT: 40
REFERENCE(S):
(1) Akaike, T; Arch Biochem Biophys 1992, V294, P55 CAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 10 OF 26 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 2000:680491 SCISEARCH
THE GENUINE ARTICLE: 350AN
TITLE: Efficient gene transfer to airway epithelium using recombinant Sendai virus
AUTHOR: Yonemitsu Y; Kitson C; Ferrari S; Farley R; Griesenbach U; Judd D; Steel R; Scheid P; Zhu J; Jeffery P K; Kato A; Hasan M K; **Nagai Y**; Masaki I; Fukumura M; **Hasegawa M**; Geddes D M; Alton E W F W (Reprint)
CORPORATE SOURCE: UNIV LONDON IMPERIAL COLL SCI TECHNOL & MED, SCH MED, NATL HEART & LUNG INST, DEPT GENE THERAPY, LONDON, ENGLAND (Reprint); UNIV LONDON IMPERIAL COLL SCI TECHNOL & MED, SCH MED, NATL HEART & LUNG INST, DEPT GENE THERAPY, LONDON, ENGLAND; NATL INST INFECT DIS, DEPT VIRAL INFECT & VACCINE CONTROL, TOKYO, JAPAN; NATL INST INFECT DIS, AIDS RES CTR, TOKYO, JAPAN; KYUSHU UNIV, GRAD SCH MED SCI, DEPT PATHOL, DIV PATHOPHYSIOL & EXPT PATHOL, FUKUOKA 812, JAPAN; DNAVEC RES INC, TSUKUBA, IBARAKI, JAPAN
COUNTRY OF AUTHOR: ENGLAND; JAPAN
SOURCE: NATURE BIOTECHNOLOGY, (SEP 2000) Vol. 18, No. 9, pp. 970-973.
Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707.
ISSN: 1087-0156.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; AGRI
LANGUAGE: English
REFERENCE COUNT: 25
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Clinical studies of gene therapy for cystic fibrosis (CF) suggest that the key problem is the efficiency of gene transfer to the airway epithelium. The availability of relevant **vector** receptors, the transient contact time between **vector** and epithelium, and the barrier function of airway mucus contribute significantly to this problem. We have recently developed recombinant **Sendai Virus** (**SeV**) as a new gene transfer agent. Here we show that **SeV** produces efficient transfection throughout the respiratory tract of both mice and ferrets in vivo, as well as in freshly obtained human nasal

epithelial cells in vitro. Gene transfer efficiency was several log orders greater than with cationic liposomes or adenovirus. Even very brief contact time was sufficient to produce this effect, and levels of expression were not significantly reduced by airway mucus. Our investigations suggest that **SeV** may provide a useful new **vector** for airway gene transfer.

L47 ANSWER 11 OF 26 JICST-EPlus COPYRIGHT 2001 JST
ACCESSION NUMBER: 1000302008 JICST-EPlus
TITLE: Green Fluorescent Protein Gene Insertion of Sendai Virus Infection in Nude Mice: Possibility as an Infection Tracer.
AUTHOR: AGUNGPRIYONO D R; YAMAGUCHI R; UCHIDA K; TATEYAMA S
TOHYA Y
KATO A
NAGAI Y
ASAKAWA M
CORPORATE SOURCE: Miyazaki Univ., Miyazaki, Jpn
Kagoshima Univ., Kagoshima, Jpn
National Inst. Infectious Diseases, Tokyo, Jpn
National Inst. Infectious Diseases, Tokyo, Jpn
Dnavec Res. Inst., Ibaraki, Jpn
SOURCE: J Vet Med Sci, (2000) vol. 62, no. 2, pp. 223-228.
Journal Code: F0905A (Fig. 4, Tbl. 1, Ref. 24)
ISSN: 0916-7250
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Short Communication
LANGUAGE: English
STATUS: New
AB The green fluorescent protein(GFP) marker from jellyfish Aequorea victoria is considered to have potential use in the study of host-pathogen relationships, by tracing infections in living cells, organs and animals. We compared the pathogenicity of Sendai virus with an inserted GFP gene(GFP-**SeV**) with that of its wild-type(Wt-**SeV**) to determine the usefulness of the recombinant virus in long-term infection of BALB/c nude(nu/nu) mice. The results indicated that the presence of GFP in infected cells could be analyzed easily and sensitively. GFP helped in identifying and in understanding the cellular sites of viral replication in vitro and in vivo. However, the GFP insertion into the Wt-**SeV** genome, led to decreased pathogenicity, altering the in vivo viral kinetics. (author abst.)

L47 ANSWER 12 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 9
ACCESSION NUMBER: 1999:675874 CAPLUS
DOCUMENT NUMBER: 132:45698
TITLE: Sendai virus gene start signals are not equivalent in reinitiation capacity: moderation

at the fusion protein gene

AUTHOR(S): Kato, Atsushi; Kiyotani, Katsuhiro; Hasan, Mohammad K.; Shioda, Tatsuo; Sakai, Yuko; Yoshida, Tetsuya; **Nagai, Yoshiyuki**

CORPORATE SOURCE: Department of Viral Infection, Institute of Medical Science, University of Tokyo, Tokyo, 108-8639, Japan

SOURCE: J. Virol. (1999), 73(11), 9237-9246
CODEN: JOVIAM; ISSN: 0022-538X

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In paramyxovirus transcription, viral **RNA** polymerase synthesizes each monocistronic mRNA by recognizing the gene start (S) and end (E) signals flanking each gene. These signal sequences are well conserved in the virus family; nevertheless, they do exhibit some variations even within a virus species. In Sendai virus (**SeV**) Z strain, the E signals are identical for all six genes but there are four (N, P/M/HN, F, and L) different S signals with one or two nucleotide variations. The significance of these variations for in vitro and in vivo replication has been unknown. We addressed this issue by **SeV** reverse genetics. The luciferase gene was placed between the N and P gene so that recombinant **SeVs** expressed luciferase under the control of each of the four different S signals. The S signal for the F gene was found to drive a lower level of transcription than that of the other three, which exhibited comparable reinitiation capacities. The polar attenuation of **SeV** transcription thus appeared to be not linear but biphasic. Then, a mutant **SeV** whose F gene S signal was replaced with that used for the P, M, and HN genes was created, and its replication capability was examd. The mutant produced a larger amt. of F protein and downstream gene-encoded proteins and replicated faster than wild-type **SeV** in cultured cells and in embryonated eggs. Compared with the wild type, the mutant virus also replicated faster in mice and was more virulent, requiring a dose 20 times lower to kill 50% of mice. On the other hand, the unique F start sequence as well as the other start sequences are perfectly conserved in all **SeV** isolates sequenced to date, including highly virulent fresh isolates as well as egg-adapted strains, with a virulence several magnitudes lower than that of the fresh isolates. This moderation of transcription at the F gene may therefore be relevant to viral fitness in nature.

REFERENCE COUNT: 42

REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 13 OF 26 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:799535 SCISEARCH
THE GENUINE ARTICLE: 246JZ
TITLE: Double-layered membrane vesicles released from mammalian cells infected with Sendai virus expressing the matrix protein of vesicular stomatitis virus
AUTHOR: Sakaguchi T (Reprint); Uchiyama T; Fujii Y; Kiyotani K; Kato A; **Nagai Y**; Kawai A; Yoshida T
CORPORATE SOURCE: HIROSHIMA UNIV, SCH MED, DEPT BACTERIOL, MINAMI KU, 1-2-3 KASUMI, HIROSHIMA 7348551, JAPAN (Reprint); UNIV TOKUSHIMA, SCH MED, DEPT VIROL, TOKUSHIMA 7708503, JAPAN; KYOTO UNIV, DEPT PHARMACEUT SCI, KYOTO 6068304, JAPAN; UNIV TOKYO, INST MED SCI, DEPT VIRAL INFECT, TOKYO 1088639, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: VIROLOGY, (10 OCT 1999) Vol. 263, No. 1, pp. 230-243

Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0042-6822.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 27

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The matrix (M) protein of vesicular stomatitis virus (VSV) was reported to form vesicles on the cell surface and subsequently to be released into the cultured medium when expressed from cDNA by virus **vectors**. To further investigate VSV M activity, we generated a recombinant **Sendai virus** (**SeV**) expressing the VSV M protein (**SeV**-M-VSV). When cells were infected with **SeV**-M-VSV, VSV M was found abundantly in the culture medium. Electron microscopy demonstrated the budding of two-membraned vesicles (greater than or equal to 0.8 mu m in diameter) from the infected cells. The outer membrane of the vesicle was derived from the plasma membrane and the inner one possibly derived from the membrane of an intracellular vesicle. Immune-gold labeling showed that VSV M was exclusively located in a double-layered region. The released membranes were divided into three parts: the VSV M Vesicles with **SeV** F and HN glycoproteins, **SeV** particles, and vesicles associated with the cytosolic components. The last abundantly contained phosphorylated **SeV** matrix (M) protein, which is not

released in a usual **SeV** infection. Furthermore the VSV M protein expressed without using a virus **vector** was efficiently released into the culture medium. These results suggest that the VSV M protein has a budding activity per se and that **SeV** proteins are passively involved in the release of VSV M. (C) 1999 Academic Press.

L47 ANSWER 14 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 10
ACCESSION NUMBER: 1999:483920 CAPLUS
DOCUMENT NUMBER: 131:282150
TITLE: Accommodation of foreign genes into the Sendai virus genome: sizes of inserted genes and viral replication
AUTHOR(S): Sakai, Yuko; Kiyotani, Katsuhiro; Fukumura, Masayuki; Asakawa, Makoto; Kato, Atsushi; Shioda, Tatsuo; Yoshida, Tetsuya; Tanaka, Akemi; **Hasegawa, Mamoru; Nagai, Yoshiyuki**
CORPORATE SOURCE: Institute of Medical Science, Department of Viral Infection, University of Tokyo, Tokyo, Japan
SOURCE: FEBS Lett. (1999), 456(2), 221-226
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

Please find most recent publication

AB Sendai virus (**SeV**) is an enveloped virus with a neg. sense genome **RNA** of about 15.3 kb. We previously established a system to recover an infectious virus entirely from **SeV** cDNA and illustrated the feasibility of using **SeV** as a novel expression vector. Here, we have attempted to insert a series of foreign genes into **SeV** of different lengths to learn how far **SeV** can accommodate extra genes and how the length of inserted genes affects viral replication in cells cultured in vitro and in the natural host, mice. We show that a gene up to 3.2 kb can be inserted and efficiently expressed and that the replication speed as well as the final virus titers in cell culture are proportionally reduced as the inserted gene length increases. In vivo, such a size-dependent effect was not very clear but a remarkably attenuated replication and pathogenicity were generally seen. Our data further confirmed reinforcement of foreign gene expression in vitro from the V(-) version of **SeV** in which the accessory V gene had been knocked out. Based on these results, we discuss the utility of **SeV** vector in terms of both efficiency and safety.

REFERENCE COUNT: 14
REFERENCE(S): (1) Calain, P; J Virol 1993, V67, P4822 CAPLUS
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CAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 15 OF 26 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:793179 SCISEARCH
THE GENUINE ARTICLE: 245UY
TITLE: Knockout of the Sendai virus C gene eliminates the viral ability to prevent the interferon-alpha/beta-mediated responses
AUTHOR: Gotoh B (Reprint); Takeuchi K; Komatsu T; Yokoo J; Kimura Y; Kurotani A; Kato A; **Nagai Y**
CORPORATE SOURCE: FUKUI MED UNIV, DEPT MICROBIOL, SHIMOAIZUKI 23-3, FUKUI 9101193, JAPAN (Reprint); FUKUI MED UNIV, RADIOISOTOPE RES INST, FUKUI 9101193, JAPAN; NATL INST INFECT DIS, DEPT VIRAL DIS & VACCINE CONTROL, TOKYO 2080011, JAPAN; UNIV TOKYO, INST MED SCI, DEPT VIRAL INFECT, MINATO KU, TOKYO 1088639, JAPAN; NATL INST INFECT DIS, AIDS RES CTR, SHINJUKU KU, TOKYO 1628640, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: FEBS LETTERS, (8 OCT 1999) Vol. 459, No. 2, pp. 205-210.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 44
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Sendai virus (**SeV**) renders cells unresponsive to interferon (IFN)-alpha. To identify viral factors involved in this process, we examined whether recombinant **SeVs**, which could not express V protein, subsets of C proteins (C, C', Y1 and Y2) or any of four C proteins, retained the capability of impeding IFN-a-mediated responses. Among these viruses, only the 4C knockout virus completely lost the ability to suppress the induction of IFN-alpha-stimulated gene products and the subsequent establishment of an anti-viral state. These findings reveal crucial roles of the **SeV** C proteins in blocking IFN-alpha-mediated responses. (C) 1999 Federation of European Biochemical Societies.

L47 ANSWER 16 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 11
ACCESSION NUMBER: 1998:258678 CAPLUS
DOCUMENT NUMBER: 129:38500

TITLE: Sendai virus C proteins are categorically nonessential gene products but silencing their expression severely impairs viral replication and pathogenesis

AUTHOR(S): Kurotani, Atsushi; Kiyotani, Katsuhiro; Kato, Atsushi; Shioda, Tatsuo; Sakai, Yuko; Mizumoto, Kiyohisa; Yoshida, Tetsuya; **Nagai, Yoshiyuki**

CORPORATE SOURCE: Department of Viral Infection, Institute of Medical Science, University of Tokyo, Tokyo, 108, Japan

SOURCE: Genes Cells (1998), 3(2), 111-124
CODEN: GECEFL; ISSN: 1356-9597

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The P/C mRNA of Sendai virus (**SeV**), a prototypic member of the family Paramyxoviridae in the Mononegavirales superfamily comprising a large no. of nonsegmented neg. strand **RNA** viruses, encodes a nested set of accessory proteins, C', C, Y1 and Y2, referred to collectively as C proteins, initiating, resp., at ACG/81 and AUGs/114, 183, 201 in the +1 frame relative to the ORF of phospho (P) protein, the smaller subunit of **RNA** polymerase. Among them, C is the major species expressed in infected cells at a molar ratio which is several-fold higher than the other three. However, their function has remained an enigma. It has not even been established whether or not the C proteins are essential for viral replication. Many other viruses in Mononegavirales encode C-like proteins, but their roles also remain to be defined. By taking advantage of a recently developed reverse genetics system to recover infectious **SeV** from cDNA, we created mutants in which C protein frames were variously silenced. C/C'(-) viruses which did not express C and C', but did express Y1 and Y2, were severely attenuated in replication in tissue culture cells of various species and tissues, as well as in embryonated chicken eggs. More notably, they were almost totally incapable of growing productively in-and hence nonpathogenic for mice-the natural host. Both gene expression and genome replication appeared to be impaired in C/C'(-) viruses. Addnl. silencing the Y1 and Y2 expression was also possible, and a critically impaired but viable clone, the 4C(-) virus, was isolated which expressed none of the four C proteins. **SeV** C proteins are categorically non-essential gene products, but greatly contribute to full replication capability in vitro and are indispensable for in vivo multiplication and pathogenesis. This study represents the first comprehensive functional assessment of the accessary C protein for Mononegavirales.

L47 ANSWER 17 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 12
ACCESSION NUMBER: 1998:165105 CAPLUS
DOCUMENT NUMBER: 128:304508
TITLE: Large quantity production with extreme convenience of human SDF-1.alpha. and SDF-1.beta. by a **Sendai virus vector**
AUTHOR(S): Moriya, Chikaya; Shioda, Tatsuo; Tashiro, Kei; Nagasawa, Takashi; Ikegawa, Masaya; Ohnishi, Yukano; Kato, Atsushi; Hu, Huiling; Xin, Xiaomi; Hasan, Mohammad K.; Maekawa, Midori; Takebe, Yutaka; Sakai, Yuko; Honjo, Tasuku; **Nagai, Yoshiyuki**
CORPORATE SOURCE: Minato-ku, 4-6-1 Shiroganedai, Institute of Medical Science, Department of Viral Infection, University of Tokyo, Tokyo, 108, Japan
SOURCE: FEBS Lett. (1998), 425(1), 105-111
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We describe a robust expression of human stromal cell-derived factor-1.alpha. (SDF-1.alpha.) and SDF-1.beta., the members of CXC-chemokine family, with a novel **vector** system based upon **Sendai virus**, a non-segmented neg. strand **RNA** virus. Recombinant SDF-1.alpha. and SDF-1.beta. were detected as a major protein species in culture supernatants, reached as high as 10 .mu.g/mL. This remarkable enrichment of the products allowed us to use even the crude supernatants as the source for biol. and antiviral assays without further concn. nor purifn. and will thus greatly facilitate to screen their genetically engineered derivs.

L47 ANSWER 18 OF 26 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:385709 CAPLUS
DOCUMENT NUMBER: 127:1636
TITLE: Preparation of (-)-stranded **RNA** virus vector having autonomously replicating activity
INVENTOR(S): **Nagai, Yoshiyuki**; Kato, Atsushi; Murai, Fukashi; Asakawa, Makoto; Sakata, Tsuneaki; **Hasegawa, Mamoru**; Shioda, Tatsuo
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan; Magai, Yoshiyuki; Kato, Atsushi; Murai, Fukashi; Asakawa, Makoto; Sakata, Tsuneaki; Hasegawa, Mamoru; Shioda, Tatsuo
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9716538	A1	19970509	WO 1996-JP3068	19961022
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI				
CA 2236113	AA	19970509	CA 1996-2236113	19961022
AU 9673351	A1	19970522	AU 1996-73351	19961022
EP 864645	A1	19980916	EP 1996-935402	19961022
R: AT, CH, DE, DK, FR, GB, IT, LI, NL, SE, FI				

PRIORITY APPLN. INFO.: JP 1995-308315 19951031
WO 1996-JP3068 19961022

AB A process for reconstituting virions of Sendai virus by introducing Sendai virus into a host in which early replication genes have been all expressed. The (-)-strand **RNA** virus vectors exhibits autonomous replication and cell-infection capability, but not propagation capability, are useful for the expression of heterologous gene during, e.g., gene therapy. Also claimed are (1) animal cells expressing Sendai virus genes M, NP, P/C, L, F, HN, or a subset of them; (2) methods for manufg. heterologous proteins from animal tissue culture fluid or chorioallantoic fluid; and (3) the Sendai virus **RNA** with defective gene M, F, or HN.

L47 ANSWER 19 OF 26 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:627474 CAPLUS
DOCUMENT NUMBER: 127:304493
TITLE: Importance of the cysteine-rich carboxyl-terminal half of V protein for Sendai virus pathogenesis
AUTHOR(S): Kato, Atsushi; Kiyotani, Katsuhiro; Sakai, Yuko; Yoshida, Tetsuya; Shioda, Tatsuo; **Nagai, Yoshiyuki**
CORPORATE SOURCE: Department of Viral Infection, Institute of Medical Science, University of Tokyo, Tokyo, 108, Japan
SOURCE: J. Virol. (1997), 71(10), 7266-7272
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal

LANGUAGE: English

AB The Sendai virus V protein is a nonstructural trans-frame protein whose cysteine-rich C-terminal half is fused to the acidic N-terminal half of the P protein via mRNA editing. The authors recently created a mutant by disrupting the editing motif, which is devoid of mRNA editing and hence unable to produce the V protein, and demonstrated that this V(-) virus replicated normally or even faster with augmented gene expression and cytopathogenicity in cells in vitro, but was strongly attenuated in pathogenicity for mice (A. Kato, K. Kiyotani, Y. Sakai, T. Yoshida, and Y. Nagai, EMBO J. 16:578-587, 1997). Thus, although categorized as a nonessential protein, the V protein appeared to encode a luxury function required for the viral in vivo pathogenesis. Here, the authors created another version of a V-deficient mutant, V.DELTA.C, encoding only the N-terminal half but not the V-specific C-terminal half, by introducing a stop codon in the trans-V frame, and then compared its in vitro and in vivo phenotypes with those of the V(-) and wild-type viruses. The V.DELTA.C virus was found to be similar to the wild-type virus in vitro with no augmented gene expression and cytopathogenicity, but in vivo, it resembled the V(-) virus, displaying a similarly attenuated phenotype. Thus, the pathogenicity determinant in the V protein was mapped to the C-terminal half. The N-terminal half was likely sufficient to confer normal (wild-type) in vitro phenotypes.

L47 ANSWER 20 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 13

ACCESSION NUMBER: 1997:708223 CAPLUS

DOCUMENT NUMBER: 128:30821

TITLE: Creation of an infectious recombinant Sendai virus expressing the firefly luciferase gene from the 3' proximal first locus

AUTHOR(S): Hasan, Mohammad K.; Kato, Atsushi; Shioda, Tatsuo; Sakai, Yuko; Yu, Deshan; **Nagai, Yoshiyuki**

CORPORATE SOURCE: Department of Viral Infection, Institute of Medical Science, University of Tokyo, Tokyo, 108, Japan

SOURCE: J. Gen. Virol. (1997), 78(11), 2813-2820
CODEN: JGVIAY; ISSN: 0022-1317

PUBLISHER: Society for General Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A genetic engineering approach was made to generate a recombinant non-segmented neg.-strand **RNA** virus, Sendai virus (**SeV**) of the family Paramyxoviridae, that expresses firefly luciferase. The DNA construct contg. the entire open reading frame (ORF) of the luciferase gene followed by the **SeV** transcription stop and restart signals connected with the conserved

intergenic three nucleotides was inserted immediately before the ORF of the viral 3'-proximal nucleocapsid (N) protein gene in a full-length **SeV** cDNA copy. After intracellular expression of full-length antigenomic transcripts from the engineered cDNA and of the viral nucleocapsid protein and **RNA** polymerase from the resp. plasmids, a recombinant **SeV** expressing luciferase activity at a high level was recovered, although the tendency of this particular reporter gene product to aggregate in cells made it difficult to est. the max. level of expression. The increase in genome length brought about by inserting 1728 nucleotides into the 15384 nucleotide parental **SeV** was assocd. with reduced plaque size, slightly slower replication kinetics and a severalfold decrease in yield of the virus. The inserted luciferase gene was stably maintained after numerous rounds of replication by serial passages in chick embryos. These results indicate the potential utility of **SeV** as a novel expression vector.

L47 ANSWER 21 OF 26 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 97186724 MEDLINE
DOCUMENT NUMBER: 97186724 PubMed ID: 9034340
TITLE: The paramyxovirus, Sendai virus, V protein encodes a luxury function required for viral pathogenesis.
AUTHOR: Kato A; Kiyotani K; Sakai Y; Yoshida T; **Nagai Y**
CORPORATE SOURCE: Department of Viral Infection, Institute of Medical Science, University of Tokyo, Minato-ku, Japan.
SOURCE: EMBO JOURNAL, (1997 Feb 3) 16 (3) 578-87.
Journal code: EMB; 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970321
Last Updated on STN: 20000303
Entered Medline: 19970313

AB The Sendai virus (**SeV**) V protein is characterized by the unique cysteine-rich domain in its carboxy-terminal half which is fused to the amino-terminal half of the P protein, but its function has remained enigmatic. The V protein-directing mRNA is generated by a remarkable process known as mRNA editing involving the pseudotemplated addition of a single G residue at a specific septinucleotide locus in the P gene, whereas the unedited exact copy encodes the P protein. Here, we introduced two nucleotide changes in the septinucleotide motif (UUUUCCC to UUCUUCC) in a full-length **SeV** cDNA and were able to recover a virus from the cDNA, which was devoid of mRNA editing and hence unable to synthesize the

V protein. Compared with the parental wild-type virus with regard to gene expression, replication and cytopathogenicity in various cell lines in vitro, the V(-) virus was found to be either potentiated or comparable but never attenuated. The V(-) virus, however, showed markedly attenuated in vivo replication capacity in and pathogenicity for mice. Thus, though categorized as a nonessential gene product, **SeV** V protein encodes a luxury function required for in vivo pathogenicity.

L47 ANSWER 22 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 15
ACCESSION NUMBER: 1997:644844 CAPLUS
DOCUMENT NUMBER: 127:316515
TITLE: Sendai virus-based expression of HIV-1 gp120: reinforcement by the V(-) version
AUTHOR(S): Yu, Deshan; Shioda, Tatsuo; Kato, Atsushi; Hasan, Mohammad K.; Sakai, Yuko; **Nagai, Yoshiyuki**
CORPORATE SOURCE: Department of Viral Infection, Institute of Medical Science, University of Tokyo, Tokyo, 108, Japan
SOURCE: Genes Cells (1997), 2(7), 457-466
CODEN: GECEFL; ISSN: 1356-9597
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We have established a system for recovering Sendai virus (**SeV**), a nonsegmented neg. strand **RNA** virus, entirely from cDNA at an extremely high rate, and have succeeded in creating a V(-) **SeV** whose gene expression was greatly enhanced by the deletion of the nonessential V gene. Because of its extreme medical importance, there has been a strong need for the establishment of a better system to express the gp120 envelope glycoprotein of the human immunodeficiency virus type 1 (HIV-1) in sufficient quantity and purity. It also remains to be established to produce gp120 in in vitro natural host cells for HIV-1 such as human primary blood mononuclear cells, macro-phages or established T cell lines. Using the above system, we created recombinant Sendai viruses expressing the gp120 in CV1 cells, a monkey kidney line. The expression level from the std. V(+) version has already reached 2.2.mu.g per 106 infected cells, which was readily purified from the culture fluid with a recovery rate of about 60%, and has so far appeared to be functionally and serol. authentic. The inserted gp120 gene was stably maintained during numerous passages of the recombinant virus. The V(-) version-based expression was even more robust, consistently reaching over 6.0 .mu.g per 106 cells, a level that is one of the highest currently attainable for gp120 prodn. in mammalian cells. Furthermore, a broad host range of **SeV** allowed gp120 prodn. in all the three natural host cells for HIV-1

described above. Se V-based expression serves as a novel choice for producing large quantities of HIV-1 gp120 and will greatly facilitate biochem., biol. and immunol. studies of this important glycoprotein.

L47 ANSWER 23 OF 26 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 16
ACCESSION NUMBER: 1997:622299 CAPLUS
DOCUMENT NUMBER: 127:290408
TITLE: Phosphorylation of the Sendai virus M protein is not essential for virus replication either in vitro or in vivo
AUTHOR(S): Sakaguchi, Takemasa; Kiyotani, Katsuhiro; Kato, Atsushi; Asakawa, Makoto; Fujii, Yutaka; **Nagai, Yoshiyuki**; Yoshida, Tetsuya
CORPORATE SOURCE: Department of Bacteriology, Hiroshima University School of Medicine, Hiroshima, 734, Japan
SOURCE: Virology (1997), 235(2), 360-366
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A large proportion of intracellular Sendai virus (**SeV**) M proteins is phosphorylated, but in mature virions the M protein is not phosphorylated or dephosphorylated. Phosphorylated M protein in cells in bound to the cytoskeletal components more firmly than unphosphorylated M protein. Thus, it has been hypothesized that M protein phosphorylation plays an important role in the virus life cycle, esp. in the step of maturation. Here, a transient expression-mutation expt. of the M gene demonstrated that a change of the Ser residue at the 70th position from the N-terminus to Ala (S70A) totally abolished M protein phosphorylation, strongly suggesting that this residue is phosphorylated. The mutated M gene was then placed in the corresponding region in the cDNA plasmid which generates a full-length antigenome **SeV RNA**, and a mutant **SeV** M-S70A was successfully recovered from the cDNA. This mutant virus was indeed defective in M protein phosphorylation but did not differ at all from the wild-type **SeV** recovered from the parental cDNA either in the replication kinetics and plaque morphol. in cultured cells or in in vivo replication and pathogenicity for mice. No phosphorylation of the M protein apparently was required for **SeV** replication either in vitro or in vivo.

L47 ANSWER 24 OF 26 JICST-EPlus COPYRIGHT 2001 JST
ACCESSION NUMBER: 990254753 JICST-EPlus
TITLE: Negative Strand **RNA** Virus, **Sendai Virus, Vector**; Regulation and Expression of the Foreging Gene.

AUTHOR: KATO ATSUSHI; YU D; HASAN M K; SAKAI(TAGAWA) YUKO; SHIODA TATSUO; **NAGAI YOSHIYUKI** KIYOTANI KATSUHIRO; YOSHIDA TETSUYA
CORPORATE SOURCE: Univ. of Tokyo
Hiroshima Univ., Sch. of Med.
SOURCE: Nippon Bunshi Seibutsu Gakkai Nenkai Puroguramu, Koen Yoshishu, (1997) vol. 20th, pp. 205. Journal Code: L1278A
PUB. COUNTRY: Japan
LANGUAGE: Japanese
STATUS: New

L47 ANSWER 25 OF 26 JICST-EPlus COPYRIGHT 2001 JST
ACCESSION NUMBER: 980583001 JICST-EPlus
TITLE: Large quantity production with extreme convenience of human SDF-1.ALPHA. by a **Sendai virus vector**.
AUTHOR: SHIODA T; KATO A; MATSUSHIMA K; **NAGAI Y** HORI T; TASHIRO K; IKEGAWA M; HONJO T NAGASAWA T
CORPORATE SOURCE: Univ. Tokyo, Tokyo, JPN
Kyoto Univ. Kyoto, JPN
Osaka Medical Center for Maternal and Child Health, Osaka, JPN
SOURCE: AIDS Res Newsl, (1997) vol. 11th, pp. 167. Journal Code: L2585A
PUB. COUNTRY: Japan
DOCUMENT TYPE: Conference; Short Communication
LANGUAGE: English
STATUS: New

L47 ANSWER 26 OF 26 JICST-EPlus COPYRIGHT 2001 JST
ACCESSION NUMBER: 970577573 JICST-EPlus
TITLE: Research on the methods to prevent the infection and occurrence. Research on the efficient expression of SDF-1a using the **Sendai virus vector**. (Ministry of Health and Welfare S).
AUTHOR: SHIOTA TATSUO; KATO ATSUSHI; MORIYA TOMOKUSA; **NAGAI YOSHIYUKI** NAGASAWA TAKESHI
CORPORATE SOURCE: Inst. of Med. Sci., Univ. of Tokyo
Osaka Medical Center and Res. Inst. Maternal and Child Health
SOURCE: Kansen Soshi oyobi Hassho Soshi Hoho no Kaihatsu ni kansuru Kenkyuhan Kenkyu Hokokusho. Heisei 8 Nendo, (1997) pp. 19-21. Journal Code: N19971434 (Ref. 7)
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article

LANGUAGE: Japanese
STATUS: New

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